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ALLERGY AND TUBERCULOUS TRACHEOBRONCHITIS

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INTRODUCTION

THE subjects which are mentioned in the title have many obscure aspects. Allergy itself is controversial. The differences between tuberculous and nontuberculous allergies are not well established. General and local tissue responses to various antigens of tubercle bacilli have been reported, but not completely correlated.

Tuberculous tracheobronchitis has been regularly recognized only in the past few years. It now appears to be very important clinically. Its occurrence, mode of involvement, types, intensity, and causative factors are still being investigated and reported. Certain points are not yet clear, including possible reasons for its selective occurrence.

A relationship between allergy and tuberculous bronchitis has been rarely and only speculatively mentioned in the literature. No proof of a connection has been reported. In spite of this, desensitization by tuberculin has been attempted. Such a method cannot be called logical, since in addition to lack of etiologic connection, no satisfactory results have been obtained from desensitization in experimental or clinical tuberculosis.

The thoracic service of the Wisconsin General Hospital is largely composed of tuberculous patients. Between 75 and 100 individuals are examined by bronchoscopy each year to confirm, exclude, or treat tuberculous tracheobronchitis. About three years ago it was observed that a number of the patients who were found to have tracheobronchial

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lesions had clinical evidence of an allergic diathesis. A scanning of the case records for the previous three years seemed to confirm this impression, and it was decided to make a thorough and precise investigation of subsequent cases.

The report of this study will be brief and not include the extensive charts, since they have shown only a few points of importance.

THE LITERATURE

To conserve further space, the publications on allergy and tuberculous bronchitis will not be minutely reviewed. References concerning allergy, including that of tuberculosis, are generally available. References on tuberculous tracheobronchitis have been summarized through the years by Eloesser, Barnwell and his associates, Samson, Ornstein, Epstein, and others. References on the relationship of allergy to tuberculous tracheobronchitis are so scanty as to be of no value. Personal communication with ten physicians in New York, Michigan, Wisconsin, and California, who have been interested in the subjects, has given no information of use; their cases were few, the studies incomplete, or the methods unsatisfactory.

There are a few conclusions from the literature which may help to form a background for the present report.

1. Asymptomatic "normal" individuals often have significant skin test reactions to the conventional allergens. Patients with tuberculosis may similarly be sensitive.

2. Sensitivity to tuberculin has scant relationship to the type, volume, or location of tuberculosis (excluding early primary and overwhelming disease).

3. Desensitization by nontuberculous antigens is not harmful to tuberculosis and has not proved of special value in cases with tuberculous bronchitis.

4. Symptoms, and the physical and x-ray signs of tuberculous bronchitis, are often helpful in suggesting a diagnosis of tuberculous tracheobronchitis.

5. The findings at bronchoscopy are highly diverse. They may include any of the tuberculous lesion types seen in other tissues.

METHODS

One hundred patients suspected of having tuberculous tracheobronchitis were bronchoscoped at least once. All examinations were made by one person (Gale).

An attempt was made to decide the presence or absence of a tuberculous tracheal or bronchial lesion, its location, its qualities (injection, edema, granulation, ulceration, fibrostenosis, or a combination), and the relative extent of the lesions. A retrograde bronchoscope was not used.

A routine history, physical examination, complete blood count (including eosinophiles), sedimentation rate, chest x-ray, and sputum and gastric concentration studies for tubercle bacilli were obtained in all cases. Smears from the bronchi of a few cases were examined for eosinophiles. The x-rays were scanned for the presence of a tension cavity, an atelectasis, or a dominantly truncal lesion. A special history was then taken for the presence of a personal or family history of asthma, hay fever, allergic rhinitis, migraine, food sensitivity, or urticaria and other suggestive skin lesions.

Skin tests were made on all patients able to remain long enough in the hospital for this purpose.

(a) Scratch and intradermal tests were made with the usual antigens, the number ranging from 120 to 160 in various cases, with routine immediate and delayed readings.

(b) Tuberculin tests were made intradermally with old tuberculin in two concentrations (0.01 and 1.0 mg.), with readings at forty-eight hours, and the reactions classified from 0 to 4 plus.

ANALYSIS OF DATA

Of the 100 patients in whom bronchoscopy was used, 80 had some evidence of tuberculous tracheobronchitis. Seventy-five per cent of these were women. (The percentage of women in the ward at that time was 53.)

Of the eighty patients with bronchial lesions, fifty were studied completely (including all skin tests) and analyzed. Eighty-two per cent of these were women (65 per cent of the patients without bronchial lesions were women). This high sex incidence has previously been reported. The men averaged 32 years of age, the women 29 years.

A correlation of the data obtained gave the following information.

1. There was a slight direct correlation of a personal history of allergy and the presence of tuberculous bronchitis by bronchoscopy. There was a tendency for small lesions to be associated with a negative history of allergy, old and fibrostenotic lesions to be associated with a positive history. Forty-five per cent of the women, and 30 per cent of the men with tuberculous bronchitis had a personal history of allergy. Only 20 per cent in groups of both men and women without bronchitis had such a history.

2. All the patients reacted to tuberculin. Sixty per cent of those with tuberculous bronchitis reacted to the more dilute dose. Seventy-two per cent of those without bronchitis reacted to the dilute dose. Most of the reactions were only 1 or 2 plus in each group.

The presence of a personal history of allergy was not associated with any special reaction to tuberculin.

3. There was a definite correlation between the presence of symptoms, signs, x-ray evidence of tuberculous bronchitis, as well as tuber-

culous laryngitis, and the presence of tuberculous tracheobronchitis by bronchoscopy. Their incidence was doubled in the cases with bronchitis. The tendency was less, but present, in regard to infectiousness. Eosinophilia was not related to the presence of bronchial lesions and eosinophiles were not prominent in smears from bronchial secretions in such cases.

4. The amount of reactivity to the conventional allergens was difficult to reduce to figures, but the general tendency and the total positiveness (1 to 4 plus) were charted, and both agreed.

In general, the occurrence and degree of reaction of the routine allergen skin tests were associated with the presence of a personal history of allergy. It was notable that most of the patients with a negative history of allergy reacted to some of the allergens.

5. There was a direct correlation between reaction to routine allergen skin tests and the skin-test reaction to tuberculin in cases with tuberculosis. Those most sensitive to tuberculin were twice as reactive to other allergens.

There was no correlation between the reaction to routine allergens and the number of eosinophiles in the blood, the presence of symptoms, x-ray or physical signs in the cases with tuberculous bronchitis. Those patients with infectious cases were more allergic than the noninfectious, but the numbers were few.

6. Patients with tuberculous bronchitis were significantly more allergic to routine allergens than were those without tuberculous bronchitis. (The average amount of skin-test reactions was twice as great.) The degree of allergy did not correlate well with the type of bronchial lesion present.

7. Eleven of the patients were found to have very extensive and serious lesions, usually involving trachea as well as bronchi. Eight of these (73 per cent) also were severely, clinically allergic. The most severe lesions occurred almost entirely in women.

COMMENTS

The cases which could be completely studied are still too few for optimal analysis.

There are several variables of such importance that they impair interpretation of the results.

1. The family and personal history of allergy is at times vague and uncertain (this is especially so for migraine, food sensitivity, and asthma in patients with pulmonary disease).

2. The significance of positive skin-test reactions to routine allergens in asymptomatic individuals is still debatable. There is a paucity of data on such "normal" controls in the literature.

3. Old tuberculin may not be the allergen which best indicates the relative allergic state of the tuberculous patient or bronchial tissues.

4. Bronchoscopy is not perfectly diagnostic, due to limited range of vision and difficulty of interpretation.

CONCLUSIONS

The deductions from the data must, therefore, be limited to the following points:

1. Patients with tuberculous tracheobronchitis reacted more strongly to routine allergen skin tests than did tuberculous patients without such lesions.

2. Tuberculous tracheobronchitis occurred more frequently in women than in men. The most serious bronchial lesions occurred predominantly in women patients with clinical evidences of allergy.

3. Patients with tuberculous tracheobronchitis were not unusually reactive to tuberculin by skin test. However, those who were most sensitive to tuberculin were most sensitive also to the routine allergens by skin test.

4. There was a general correlation between a personal history of allergy and a positive reaction to routine allergens, although most patients with a negative personal history reacted to some allergens. There was an apparent direct relationship between a personal history of allergy and the presence of tuberculous tracheobronchitis.

5. Clinical evidence of tuberculous tracheobronchitis correlated well with the finding of bronchial lesions by bronchoscopy; eosinophilia did not. Reaction to the routine allergens did not correlate well with clinical signs.

6. Attempts to desensitize patients with tuberculous tracheobronchitis, using tuberculin, do not seem indicated. Attempts to control an allergic process by desensitization to any pertinent routine allergens, or by other effective means, seem legitimate and indicated.

HUGE INTRATHORACIC FIBROMA: REPORT OF A CASE

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PRIMARY intrathoracic neoplasms occur frequently. With the employment of modern diagnostic methods they are recognized earlier and in a larger number of cases than formerly and, because of advances in anesthesia, types of surgical technique, and postoperative care, they are successfully removed surgically in an ever-increasing number of instances.

The most important and most frequent intrathoracic tumors, according to Blades,[1] are of pulmonary or bronchiogenic origin. Second in frequency are mediastinal tumors, and least frequent or comparatively rare are the neoplasms arising from the wall of the thorax and extending into the thoracic cavity. As Blades has indicated, it is difficult to determine in many instances whether an intrathoracic tumor arises from the mediastinum or from the thoracic wall, particularly when the growth is a large one and is densely adherent to both these structures.

Intrathoracic fibroma is an extremely rare tumor. Blades found only thirty-two cases of mediastinal fibroma in the world literature up to October, 1941, and Harper,[2] in 1939, was able to find only nine recorded instances of intrathoracic fibroma arising from the thoracic wall.

We wish to report the successful surgical removal of a huge intrathoracic fibroma which had no definite point of origin either from the mediastinum or from the thoracic wall, but which was densely adherent to both these structures. We believe that this fibroma is the largest solid intrathoracic tumor that ever has been removed surgically with survival and complete recovery of the patient.

REPORT OF CASE

An unmarried farmer, 44 years old, of German extraction, registered at the Mayo Clinic on Nov. 13, 1942. There was no history of inheritable familial disease; his father had died of a perforated peptic ulcer and his mother had died of pulmonary embolus following cholecystectomy. He had seven siblings, all in good health. One of his grandparents suffered from asthma. In 1920, the patient had had postinfluenzal pleurisy which had subsided spontaneously, and, in 1929, he had had polyarthritis. The only surgical procedure that he had undergone was a tonsillectomy in 1929.

The primary complaint for which he sought care at the clinic was shortness of breath, which he first had noticed only on exertion two years

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previously, in 1940. This condition had been progressive, especially during the winter of 1941. During the summer of 1942, a morning cough developed. This had produced about ½ ounce (15 c.c.) of white, thick mucus but never any blood. He never had coughed during the day. For the past few months before admission, walking the length of one block had precipitated dyspnea with wheezing, and palpitation with dull precordial pain. He had had frequent night sweats but no fever even with the frequent colds he had experienced. For one year he had noticed engorgement of the veins of his arms and neck and a purple color of his face.

The positive findings on physical examination were as follows. The systolic blood pressure was 135 and the diastolic 95 mm. of mercury; the pulse rate was 84 beats per minute. Temperature was 98.8° F.

There was nodular enlargement of the thyroid gland, grade 2 to 3, on the basis of 1 to 4. The veins of the arm and neck were greatly dilated and the patient's face was very florid. The left border of the shifted heart was found by percussion to lie at the anterior axillary line and the right border was situated to the left of the left sternal margin. There was an occasional premature beat and a systolic murmur was heard at the cardiac apex. The patient's trachea also was shifted to the left. The thorax was of the emphysematous type. There were dullness to percussion, a diminished fremitus and absent breath sounds on the right, extending over the entire thorax from the apex downward posteriorly, and below a line drawn from the acromial process to the xiphoid process anteriorly. Respiratory excursion lagged on the right side. There was moderate tenderness over the right upper abdominal quadrant and the area of liver dullness extended four fingerbreadths below the right costal margin. The patient had bilateral inguinal hernia. Clubbing of fingers and toes was very marked. Laryngoscopic examination was negative.

The flocculation test for syphilis was negative. The leucocyte count was 9,900 and the erythrocyte count was 5,190,000. The value for hemoglobin was 15.3 Gm. per 100 c.c. of blood. The initial urinalysis revealed only a few erythrocytes, and repeated tests did not reveal any albumin, sugar, casts, or cells. The basal metabolic rate was +47 per cent. The sedimentation rate was 3 mm. in one hour. Examination of the sputum for *Mycobacterium tuberculosis* was negative.

Stereoscopic roentgenograms of the thorax revealed that a large tumor filled the lower three-fourths of the right pleural cavity and forced the heart and the mediastinum to the left (Fig. 1). The mass did not extend quite to the anterior or posterior wall of the thorax. The roentgenoscopic examination of the thorax was reported as follows: "There is an extremely large, fairly well circumscribed tumor occupying the posterior two-thirds of the right thoracic cavity. The lower limits of the tumor extend across the midline behind the heart. The esophagus is markedly displaced to the left and anteriorly."

After medical and surgical consultation, it was decided that thyroidectomy for a hyperfunctioning adenoma of the thyroid should be performed first since it would greatly reduce the risk of further operation without carrying a high risk itself. Therefore, the patient was prepared for thyroidectomy by administration of compound solution of iodine (Lugol's solution). On Nov. 27, 1942, a double resection of the thyroid gland with removal of the isthmus and pyramidal lobe was performed. The left lobe was three times and the right lobe was four times the normal size. The right inferior thyroid artery was ligated. The postoperative convalescence was uncomplicated. The basal metabolic rate on Dec. 12, 1942, was +35 per cent—somewhat reduced but still elevated—very probably due to the disturbed respiratory function. The patient did not re-enter the hospital for thoracotomy until after the Christmas season, namely, on Jan. 5, 1943.

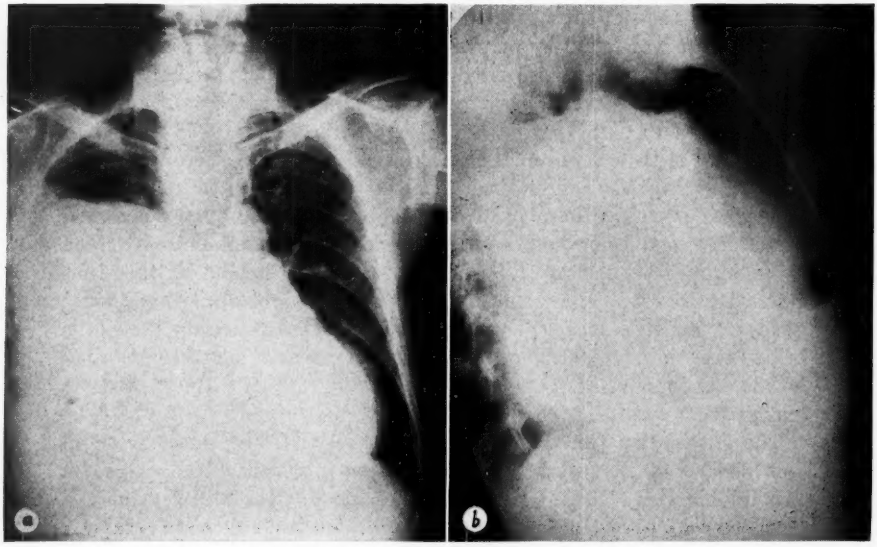

Fig. 1.—Huge tumor of right thoracic cavity which forced the heart and mediastinum to the left; *a*, anteroposterior view; *b*, lateral view.

Erythrocytes, grade 3, on a basis of 1 to 4, were discovered on urinalysis at this time, but excretory urograms and extensive urinary investigation revealed no cause for the hematuria other than a moderately enlarged prostate gland. It therefore was decided that neither operation nor preoperative prophylactic chemotherapy was contraindicated and 2 Gm. of sulfadiazine were given to the patient orally on Jan. 12, and 5 Gm. were administered on the following day.

On Jan. 14, 1943, cyclopropane, oxygen, ether, carbon dioxide, and nitrous oxide were administered intratracheally by the carbon dioxide

absorption technique and thoracotomy was performed. A long segment of the right seventh rib and segments of the sixth and eighth ribs just posterior to angle, between the angle and the transverse processes, were resected for adequate exposure of a huge tumor in the right lower part of the thorax. The tumor filled three-fourths of the right side and a third of the left side of the thorax. Biopsy was performed and the mass was reported to be fibroma and so it was decided to attempt excision. The tumor had no definite point of origin but was apparently parasitic. It was densely adherent over the entire surface to the mediastinum, thoracic wall, and collapsed lung. One large projection extended behind the pericardium and aorta and into the left side of the thorax.

Removal of the tumor caused numerous bronchopleural fistulas on the surface of the lung as it was impossible to carry out the dissection extrapleurally. These were sutured and closed as thoroughly as possible and the lung, which had been compressed for a long time, was re-expanded as completely as possible. A large intercostal tube for siphon drainage was left in the ninth interspace at the posterior axillary line. Postoperative bronchoscopy was performed to remove any bronchial secretion or obstruction. In the course of the operation it was necessary to administer 3,000 c.c. of citrated blood as a supportive measure.

After further examination of the entire tumor and microscopic examination of representative sections obtained from several regions, the pathologist confirmed the diagnosis of fibroma which had been made by biopsy. The tumor measured 36 by 22 by 11 cm., and weighed 10.9 pounds (4,972 Gm.) (Fig. 2). Microscopically, the tumor was composed of mature fibroblasts and contained an abundance of collagen. There were no mitotic figures or any other evidence of malignancy (Fig. 3).

The patient was placed in an oxygen tent promptly on his return to his room and at no time was he cyanotic. The venous engorgement and florid complexion disappeared immediately postoperatively. The patient responded within a few hours after his return to his room, but for thirty-six hours the blood pressure never was higher than 60 to 70 mm. of mercury, systolic, and 40 mm. of mercury, diastolic, in spite of the intravenous administration of fluids, epinephrine and coramine. The pulse rate, however, never was more than 80 to 100 beats per minute. Another transfusion of 500 c.c. of blood brought a transient rise of the systolic blood pressure to 100 mm. and the diastolic to 50 mm., but in three hours the pressure had returned to the former low levels. There was a very gradual rise of blood pressure in the next few days, and after the third postoperative day and until the patient's dismissal from the hospital the blood pressure ranged from 90 to 100 mm. of mercury, systolic, and from 50 to 60 diastolic. It was felt that the hypotension, which had none of the features of shock, was a result of release of

pressure by the tumor on the great vessels of the thorax and the consequent circulatory readjustment.

As a result of the hypotension, the urinary output for the first twelve hours after operation was 10 c.c., and then, in subsequent twenty-four hour periods, it rose to 320 c.c., 750 c.c., and 900 c.c.; thereafter, it varied from 800 to 1,600 c.c. The fluid intake for these periods totalled from 3,000 to 6,500 c.c. in twenty-four hours.

The patient was markedly confused mentally until the eighteenth postoperative day, when he had his first lucid moments. Improvement was progressive from that date. The temperature, taken rectally, never was higher than 101.6° F., and averaged 100° F. for the first three weeks, after which it remained normal.

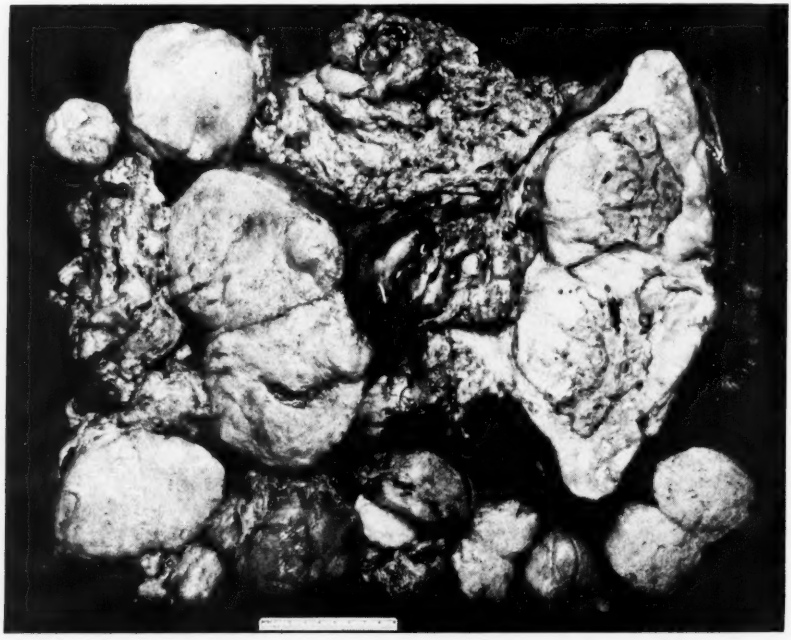

Fig. 2.—Thoracic fibroma removed in fragments (×130).

The intercostal drainage tube was removed on the seventh postoperative day. The amount of drainage in twenty-four hours during this period ranged from 100 to 1,650 c.c.

The patient was out of bed on the tenth postoperative day. He had moderate hemoptysis, which was attributed to residual bronchopleural fistulas. It was necessary on the twenty-seventh postoperative day to provide for open drainage of the residual space left by removal of the tumor from the right side of the thorax. The right lung had expanded to fill the upper two-thirds of the right pleural cavity but a mass of

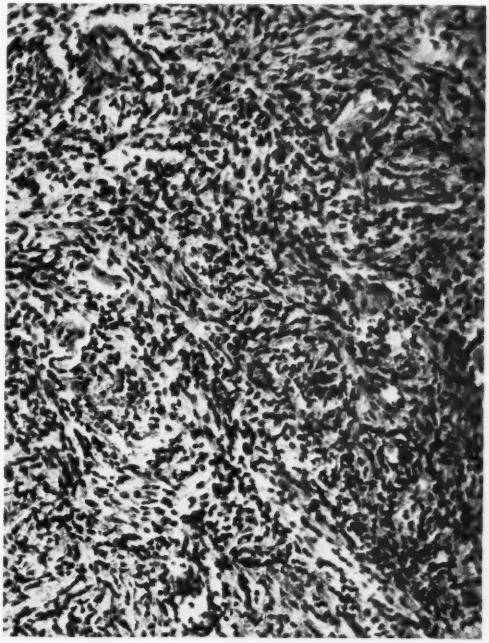

Fig. 3.—Fibroma composed of mature fibroblasts in a matrix of collagen fibers.

Fig. 4.—Roentgenologic appearance of thorax three months after operation.

clotted blood was evacuated from the lower third and the wound was left open. The wound was revised a week later for more adequate drainage and the cavity diminished rapidly in size.

When the patient was dismissed from our care on Feb. 24, 1943, forty-two days postoperatively, he was completely afebrile, well oriented, and strong. He was seen again on April 17, 1943. The thoracic wound had been irrigated with surgical solution of chlorinated soda (Dakin's solution) twice weekly in the interim. There was only a slight amount of drainage from the wound and a very small bronchopleural fistula. He had gained twenty-eight pounds (12.7 kg.) and was working on his farm. The only symptom he experienced was a very slight morning cough. Roentgenologic examination of the thorax at this time revealed some residual fibrosis and pleuritic thickening, but the lung was almost completely re-expanded (Fig. 4).

COMMENT

Benign intrathoracic tumors possess the features of all benign tumors in any portion of the body, that is, they are encapsulated and lack the invasive properties of malignant tumors. However, intrathoracic malignant tumors frequently cannot be distinguished from benign conditions by means of available diagnostic methods.

Alexander[3] said that "most circumscribed intrathoracic neoplasms are not extrapulmonary and benign, but intrapulmonary and malignant." Differential diagnosis preoperatively is possible only by means of bronchoscopy. The results of this procedure are inconclusive in about 20 per cent of cases of bronchiogenic carcinoma, according to most authors. It is apparent then that in a certain percentage of cases of intrathoracic tumors exploratory thoracotomy is the only remaining diagnostic procedure which can be offered and, in cases of operable malignant tumors, the patient's only hope.

As Overholt and Souders[4] have pointed out, exploratory thoracotomy is now fortunately almost as safe as abdominal exploration and need not be regarded as too dangerous to be warranted as a diagnostic procedure. Operation is to be recommended promptly, however, not only in cases of suspected or probable malignant lesions of the lung but also in cases in which tumors are unquestionably benign, since these lesions, by virtue of their situation in the thorax, irrespective of size, may jeopardize the patient's life because of interference with the function of one or more of the vital intrathoracic organs. Unfortunately, late surgical extirpation of these benign lesions, dangerous because of their situation, is also hazardous and sometimes impossible because of the danger of operative trauma to vital structures.

Alexander[5] and Harrington[6] stressed the danger of delaying surgical intervention of benign extrapulmonary tumors because of their propensity toward growth and consequent interference with the function of the lungs and mediastinal organs by virtue of their size alone. This point

was well demonstrated in the case which we have reported. The patient had striking signs of compression of the lung and obstruction of venous return because of the size of the mass, rather than because of its situation.

The benign intrathoracic growths may be classified as follows.

I. Anterior mediastinal tumors
   1. Lymphomas
   2. Thymomas
   3. Dermoids and teratomas
   4. Thyroid adenomas
   5. Cysts
   6. Lipomas
   7. Lymphangiomas and hemangiomas

II. Posterior mediastinal tumors
   1. Fibroma, neurofibroma, perineural fibroblastoma, and ganglioneuroma
   2. Occasional remnants of the foregut
   3. Occasional chondromas, osteomas, derived from bone and cartilage of the spine
   4. Myxomas

III. Tumors of the thoracic wall
   1. Chondroma
   2. Fibroma
   3. Osteoma
   4. "Benign" giant cell tumors
   5. Angiomas, hemangiomas, and so forth
   6. Myxomas

As has been pointed out, intrathoracic fibromas may arise either from the structures of the mediastinum or from any tissues of the thoracic wall or vertebrae. Tudor Edwards[7] was quite confident that fibromas frequently originate from tissues other than the pleura and that during the course of intrathoracic projection of these tumors the pleura is pushed ahead of them. This we could not demonstrate in our case. It may be true that some of the tumors reported in the literature as fibromas may have been derived from nerve tissue and therefore more properly diagnosed as neurofibromas, fibroblastomas, and so forth, but very careful examination of the tumor herein reported failed to reveal any indication of neurogenic origin. In this respect and as no definite point of origin could be demonstrated, this tumor very closely resembled the tumor in a case reported by Susman.[8] The fact that the tumor was a pure fibroma was a further indication for prompt surgical intervention since it is known that tumors of this type more readily undergo malignant sarcomatous change than do fibrous tumors of neurogenic origin.

Heuer and Andrus[9] have indicated that symptoms and signs of mediastinal fibromas occur in the later age groups, usually in cases in which

patients are more than 40 years of age. The usual symptoms are productive or nonproductive cough, dyspnea, and thoracic pain. Signs of a tumor are venous engorgement of the upper extremities and neck. It is unfortunate that tumors such as fibromas and neurofibromas are the most "silent" of intrathoracic neoplasms since their unwieldy size at the time the patient seeks medical advice makes the operation more hazardous. Certainly, the tumor in the case we have reported must have had its origin months or years before it manifested itself by virtue of the symptoms it caused. In spite of the latent period between origin and manifestation, surgical treatment of mediastinal fibromas has been very successful. In eighteen of the thirty-two reported cases, operation was not attempted and all the patients died. Surgical cure was effected in thirteen of the fourteen cases in which operation was performed.

The margin of respiratory safety in patients with tumors as prodigiously large as the one we have reported is extremely narrow and most of the effective aeration of the blood in the pulmonary system is accomplished through only one lung. In the period during which the tumor undergoes its insidious growth, adjustment to diminished vital capacity, pressure on the large vessels, and altered intrathoracic dynamics are made. Removal of the tumor completely upsets this adjustment, as in the case of our patient whose postoperative hypotension certainly was not true surgical shock since it failed entirely to respond to the usual measures. His disorientation and confusion too have not been satisfactorily explained on any other basis than that the effects of removal of such a huge tumor are profound. Indicative of the fact that surgical removal of the large tumors is hazardous are reports of two patients with lipomas which were larger than the fibroma in our case.[10] One patient died during infiltration of the thorax with procaine hydrochloride. At necropsy the tumor was found to measure 25 by 25 by 13 cm., and to weigh 12.43 kg. The other lipoma was partially removed, recurred, and at necropsy weighed 8.5 kg. Prompt removal of all intrathoracic neoplasms is, of course, the only way in which this difficulty may be averted.

Harrington[6, 11, 12] repeatedly has stressed the fact that roentgenographic examination is of invaluable aid to the thoracic surgeon. Routine roentgenographic examination of the thorax of every person would enable earlier diagnosis of silent tumors and would serve as well in the early detection of preclinical early tuberculous lesions.

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MEDIASTINAL LIPOMA

A Case Report

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IMPROVEMENTS in operative technique and postoperative care, and a better understanding of the physical and physiologic problems involved have markedly improved results in thoracic surgery. This is particularly true in the field of benign intrathoracic neoplasms where success is largely dependent on early diagnosis and prompt surgical intervention.

Brian Blades was able to collect 36 cases of thoracic lipoma from 1782 to 1941. They usually arise in the mediastinum and are often considered to be congenital in origin. Complete surgical removal is indicated even though the tumor is asymptomatic when diagnosed.

Three types of thoracic lipomas were described by Heuer in 1933. In the first group are those limited to the thoracic cavity, in the second group are those which extend into the neck, and in the third group, the so-called "dumbbell type," those with intrathoracic and extrathoracic portions connected by an isthmus through an intercostal space. Occasionally they reach gigantic proportions; in one instance, post-mortem examination disclosed an intrathoracic lipoma weighing 17 pounds, 6 ounces.

Signs and symptoms of intrathoracic lipoma vary according to the size and position of the tumor. A number of cases were reviewed by McCorkle and his co-workers, in 1940, and of 34 patients, 16 were dyspneic, 11 had chest pain, 7 complained of cough, 2 had hemoptysis, and 2 complained of hoarseness. In 14 instances the tumor was palpable. Chest radiographs revealed the presence of a tumor in 9 cases. Eight patients were cyanotic, 3 had edema of the extremities, 1 had an elevated diaphragm, another suffered from anginal symptoms, a third patient had cardiac arrhythmia, while still another had cardiac symptoms. Five patients were entirely asymptomatic. The patient reported by us is interesting in regard to symptomatology. In addition to the usual symptoms of dyspnea and chest pain, the chief complaint was dysphagia and epigastric pain which finally caused her to seek medical advice.

Present-day surgical treatment of benign intrathoracic neoplasms is very encouraging. Success is dependent largely upon early diagnosis and surgical intervention, while the tumor is still operable. However,

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of 34 cases reviewed by McCorkle and associates, only 12 were successfully excised. Nine patients with "dumbbell type" lipoma were operated upon, 5 being successfully excised and 4 resulting in postoperative death. Five cases of intrathoracic lipoma extending up into the neck were reported. Four of these lipomas were removed successfully, while one was found at autopsy. Nineteen cases of lipoma limited to the thorax comprised the largest group and included the largest tumors, including one which weighed 17 pounds, 6 ounces. Sixteen of these were found at autopsy. One was partially removed but later reformed and caused death by mediastinal displacement. Three were excised successfully. None of the 12 lipomas successfully excised exceeded 500 Gm. in weight.

The case being reported is particularly interesting because of the huge intrathoracic tumor present and the consequent physical and physiologic problems entailed in removing it successfully from the thoracic cavity. It is the largest mediastinal lipoma which has been excised successfully from the chest cavity and reported in the medical literature.

CASE REPORT

Mrs. R. H., a 40-year-old white woman of German descent was first seen at Memorial Hospital on Feb. 6, 1942. Two weeks previously she had been admitted to a tuberculosis sanitarium by her local doctor. Various diagnostic procedures had been carried out, a provisional diagnosis of benign intrathoracic neoplasm had been made, and she had then been referred to Memorial Hospital.

Since childhood the patient had always been below average weight. She had tired easily, and had had moderate exertional dyspnea. During the last year her dyspnea and fatigue had increased and she had experienced occasional retrosternal and epigastric pain accompanied by anorexia and rarely by nausea and vomiting. For the last three months the epigastric pain had increased markedly and the nausea and vomiting had continued, at times being accompanied by severe dyspnea. During the month prior to admission she had had increasingly severe dyspnea on slight exertion and had developed dysphagia; food particles seemed to stick in the mid-esophagus. On admission the patient complained of severe dyspnea on slight exertion, had an almost constant retrosternal and epigastric pain, and suffered from dysphagia and occasional nausea and vomiting. She had no cough, no hemoptysis, and no recent weight loss.

Review of her past history revealed that she had had a cesarean section at the age of 29. Her maximum weight had been 130 pounds, which she maintained from 1940 to 1942.

Physical examination revealed a middle-aged woman in apparent good general health. She was not dyspneic while at rest. The left hemi-

thorax was diffusely enlarged and had markedly diminished respiratory motion. In the suprasternal notch the trachea was moderately displaced to the right. The heart was also displaced to the right, its right border being percussed 8.5 cm. from the midline. Blood pressure was 120/80, there was regular sinus rhythm, and a pulse rate of 78 per minute while at rest. The right lung was clear and resonant. The left lung was flat except for a small patch superiorly and posteriorly which showed diminished resonance, and where a few moist râles and distant breath sounds were heard. Otherwise the left hemithorax was entirely flat and no voice or breath sounds were audible. There was slight fullness and some spasm in the left upper quadrant of the abdomen at the costal margin. The remainder of physical examination was within normal limits.

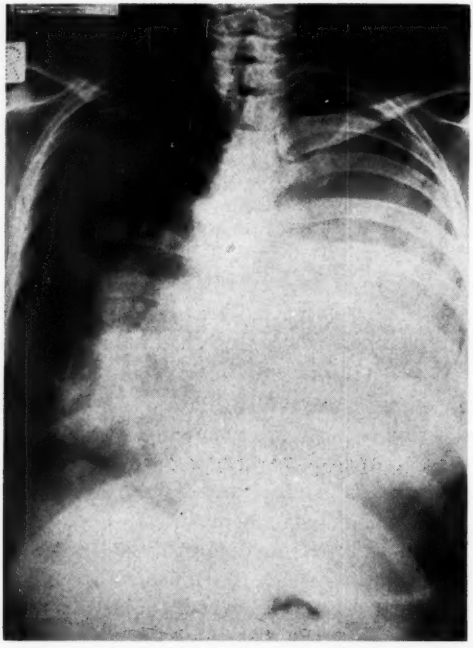


Fig. 1.—Preoperative radiograph of chest showing a large homogeneous, somewhat radiolucent mass occupying practically the entire left hemithorax. Mediastinal contents are markedly displaced to the right, left diaphragm depressed, left intercostal spaces widened.

Preoperative diagnostic procedures were carried out including chest radiographs, aspiration biopsy, contrast visualization of the mediastinal vessels, bronchospirometry, an electrocardiograph, and routine blood studies.

The chest radiograph (Fig. 1) revealed a large, ovoid, soft tissue mass almost completely filling the left hemithorax, depressing the left diaphragm, and displacing the mediastinal contents to the right. The

mass was chiefly anterior, had a well-defined border, and contained an area of increased density about 1.5 cm. in diameter in its inferomedial portion.

An aspiration biopsy was performed through the left third and fifth interspaces anteriorly in the midclavicular line. The needle passed through a firm capsule and penetrated a soft homogeneous mass to a depth of 8 cm. The aspirated material was reported by the pathologist as "fat."

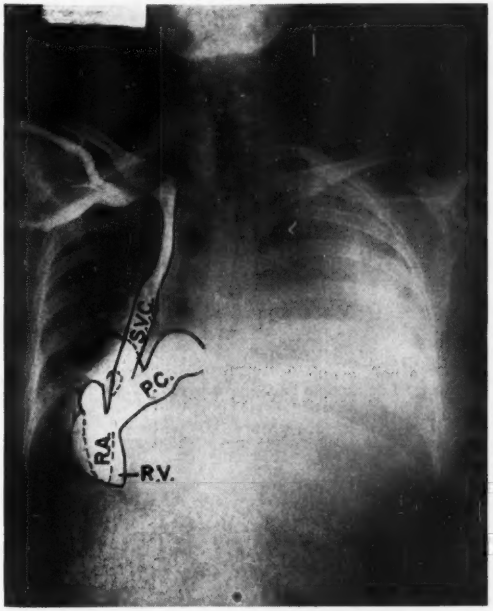


Fig. 2.—Preoperative radiograph of chest, contrast visualization, 2 seconds after injection. Dye outlines the superior vena cava, right side of heart, and the pulmonary conus.

Contrast Visualization (Figs. 2 and 3).—A 70 per cent solution of diodrast, 35 c.c., was injected into the right antecubital vein in 1.5 seconds. X-ray films were exposed at 2.0 and 4.5 seconds after beginning injection. (Circulation times were within normal limits; the arm to lung time being six seconds, and the arm to tongue time 10.5 seconds. Venous pressure in the left arm was 12.5 cm. of water). The first x-ray film with the dye outlining the superior vena cava, right side of heart, and beginning to enter the pulmonary conus revealed mediastinal shift to the right. The second film outlined the right pulmonary vessels but no dye could be visualized in the left lung field.

Bronchospirometry was performed with the bronchial catheter in the left main bronchus (Fig. 4). It revealed good ventilation and oxygen

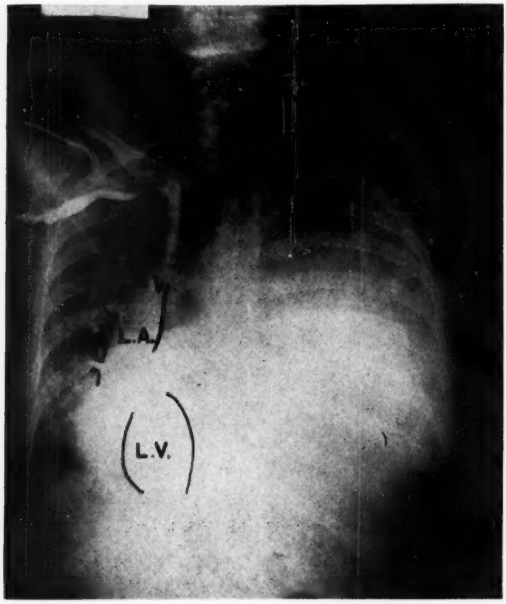


Fig. 3.—Preoperative radiograph of chest, contrast visualization, 4.5 seconds after injection. Dye entering left auricle is still present in right heart and right pulmonary vessels. Left pulmonary vessels are not visualized.

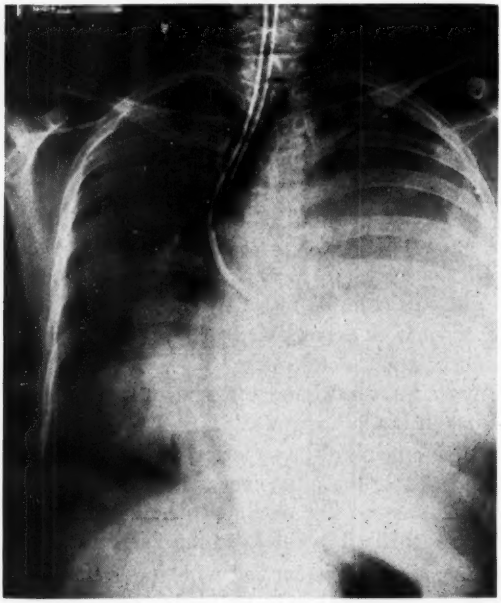


Fig. 4.—Preoperative radiograph of chest showing the bronchospirogram tube in position with the tip in the left main bronchus.

absorption by the right lung, but a markedly diminished ventilation (25 c.c.) and no oxygen absorption by the left lung (Fig. 5).

An electrocardiograph showed regular sinus rhythm, an A-V rate of 78 per minute, P-R interval of 0.16 seconds, QRS interval of 0.08 seconds. It was interpreted as a normal electrocardiogram for the position of the heart and presence of the left hemithorax mass.

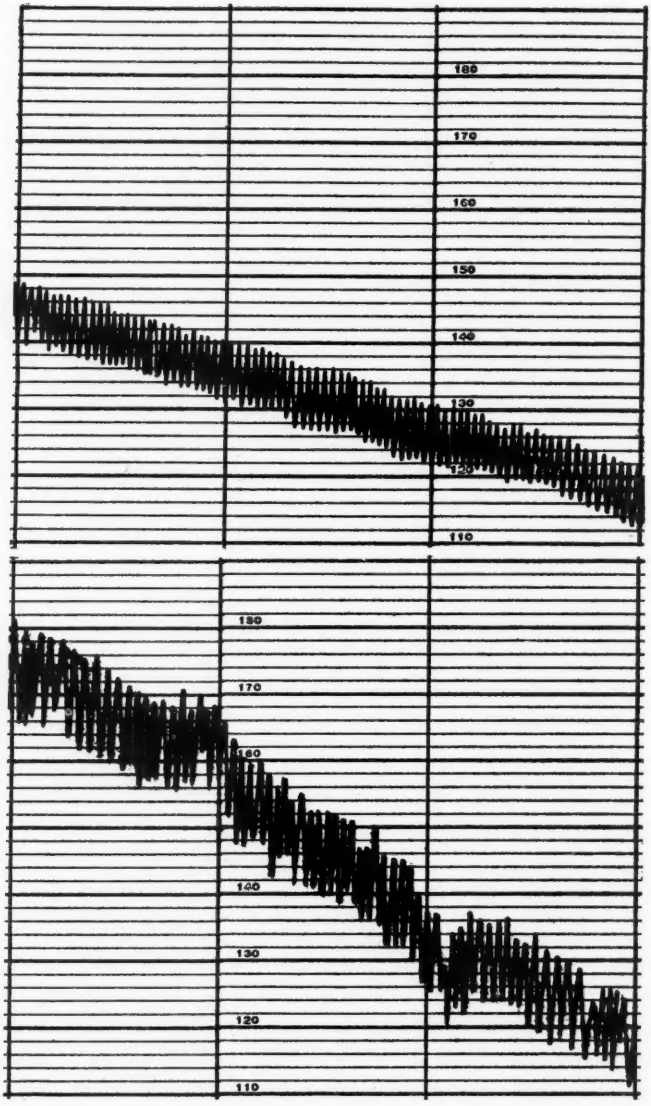
The patient's Kline test was negative. The hemoglobin was 83 to 90 per cent, red blood count 4,500,000, white blood count 4,650 to 9,000, polymorphonuclear leucocytes 61 to 75 per cent, lymphocytes 36 to 23 per cent, monocytes 1 to 2 per cent. The serum bilirubin was 1.5 mg., serum protein 7.1 mg., serum chloride 642 mg., carbon dioxide combining power 68 per cent, blood sugar 100 mg. per cent, blood urea nitrogen 6.6 mg. per cent, blood prothrombin 87 per cent.

A preoperative diagnosis of intrathoracic lipoma was established and the patient was operated upon March 3, 1942.

At operation the left hemithorax was definitely larger than the right side and moved less with respiration. When the left chest was opened, a large, well-encapsulated, ovoid tumor was found occupying the greater part of the left chest cavity, depressing the left diaphragm, and displacing the mediastinum to the right. The tumor appeared to arise from the mediastinum, its capsule which was discrete elsewhere could not be visualized medially or superiorly. The left lung was extremely compressed and atelectatic, had the appearance of liver, and occupied a small portion of the posteromedial margin of the upper half of the left hemithorax. The body of the tumor consisted of avascular firm fatty tissue. Many large veins were present in its capsule and at its base. Removal of the mass did not have any immediate effect on the position of the mediastinum (Figs. 6 and 7).

Under gas, oxygen, and ether, intratracheal anesthesia, with the patient lying on the right side (Fig. 8), the left hemithorax was entered by an oblique incision through the seventh intercostal space, extending from the angle of the scapula posteriorly to the costochondral junction anteriorly. A smooth encapsulated tumor mass presented into the wound. The wound margins were retracted and the tumor isolated anteriorly, laterally, and posteriorly by blunt dissection. No cleavage plane was found medially or superiorly where the tumor seemed to be continuous with the mediastinum. The capsule was then incised and the tumor content was evacuated piecemeal. Following this, the capsule was excised down to its reflection from the mediastinum. Hemostasis was obtained and 10 Gm. of sulfathiazole powder were dusted into the pleural cavity. The chest wall was then closed in separate layers with interrupted black silk. No drains were used. Air, 300 c.c., was aspirated from the left hemithorax, which gave a pneumothorax reading of 0, –6 (centimeters of water) at the close of the procedure. A large

A.



B.

Fig. 5.—*A*, preoperative, *B*, postoperative. Findings reveal marked improvement of both ventilation and oxygen absorption of the right lung following operation.

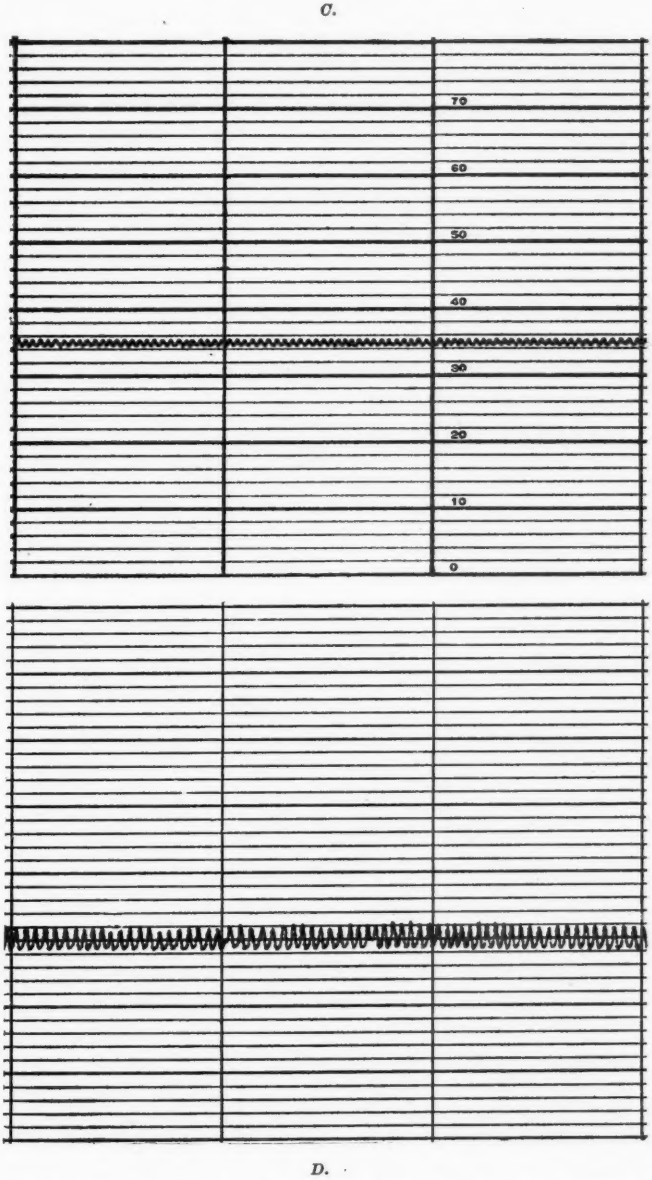


Fig. 5.—*C*, Preoperative, *D*, postoperative. The left lung, although its ventilation has increased, still does not absorb any oxygen.

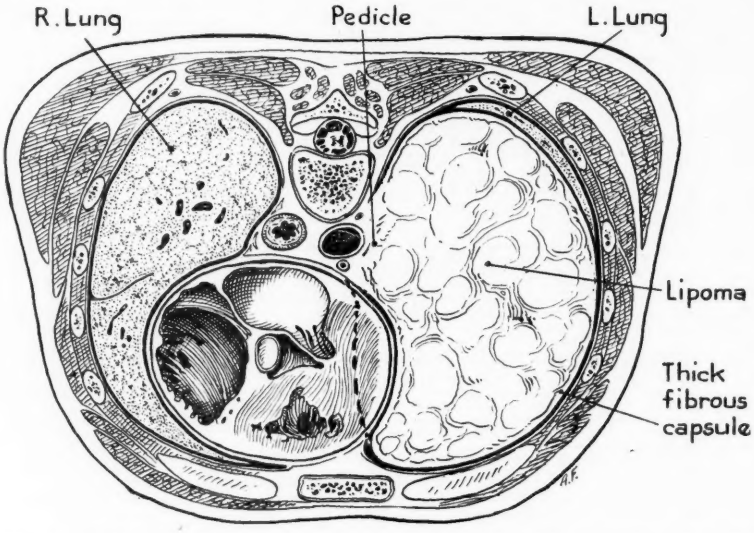


Fig. 6.—Cross section showing tumor origin in the posterior mediastinum. The heart and mediastinal contents are displaced well to the right side of chest.

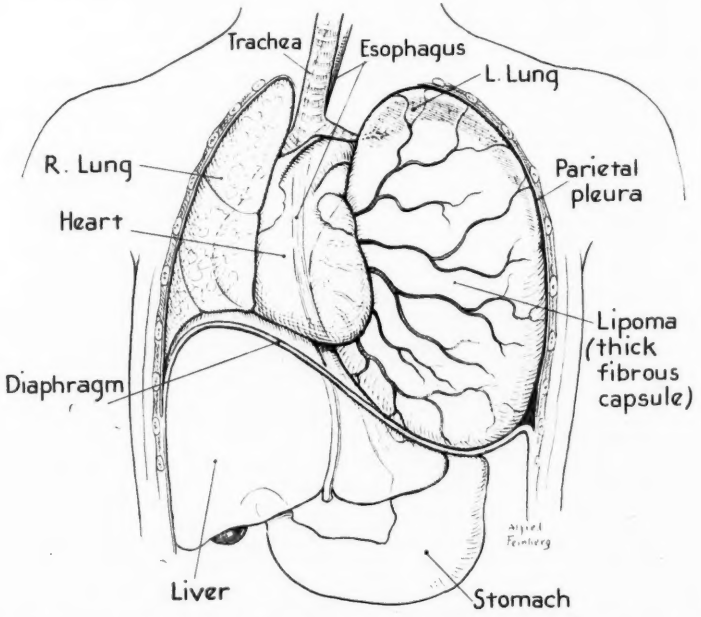


Fig. 7.—Artist's sketch showing the anatomic distortion caused by a large (6.8 pound) mediastinal lipoma. The left lung was flattened and atrophied by the tumor which practically filled the enlarged left thoracic cage.

dry dressing was applied, the patient was placed on her right side to avoid rapid mediastinal shift, and was returned to the ward in fair condition. Seven hundred cubic centimeters of blood and 300 c.c. of saline were given intravenously during the operation.

The gross specimen (Fig. 9) consisted of several large pieces of lobulated adipose tissue which together formed one large ovoid mass measuring 30 by 23 by 22 cm. One small area of calcification about 1 cm. in diameter was noted. The total weight was 3,100 Gm. or 6.8 pounds. Microscopic examination confirmed the diagnosis of lipoma.

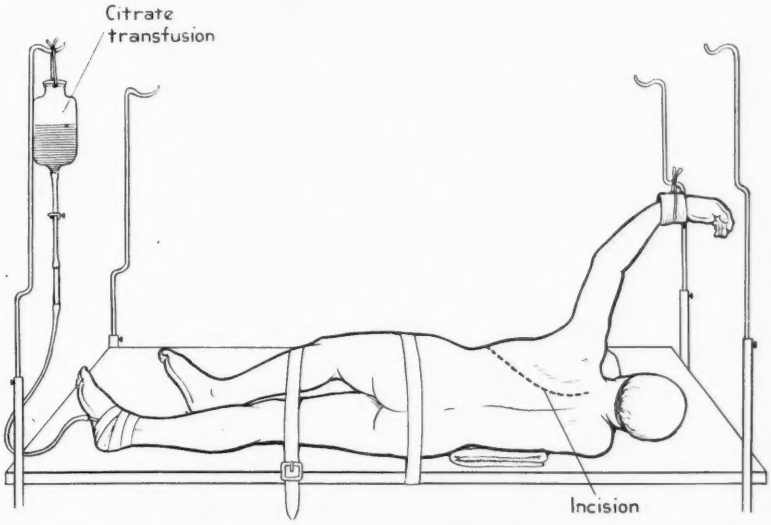


Fig. 8.—Incision for removal of a large mediastinal lipoma. The scapula is mobilized, the seventh interspace opened, and ribs spread and folded on each other by use of a modified Sauerbruch rib retractor.

On returning to the ward, the patient was placed at an angle of 45 degrees, on the right side, in an effort to avoid sudden mediastinal shift. Ten liters of oxygen were administered every minute in an oxygen tent and the infusion of 700 c.c. of blood and 500 c.c. of saline solution was slowly completed. Six hours postoperative, the patient's condition was fair with respirations 20 per minute and pulse 110 per minute and strong. However, within one hour the patient suddenly went into shock. Her pulse became weak and rose to 130 per minute, respirations were shallow and rapid, and blood pressure was not obtainable. Manifestations of pulmonary edema were present. The patient coughed up a great amount of thin frothy fluid mucous and some blood-tinged sputum. Shock blocks were placed under the foot of the bed, heat was applied to the body, coramine and an infusion of 1,500 c.c. of 10 per cent glucose in distilled water were given intravenously. Following this the patient's

condition improved. Blood pressure rose to 85/50, pulse 110, and respirations 18. Twelve hours after operation, a similar episode of shock and pulmonary edema appeared, but the patient rallied after receiving more coramine and an infusion of 250 c.c. of plasma and 250 c.c. of saline solution. Blood pressure rose to 90/60, pulse was rapid, 120 per minute but strong, and respirations only 18 per minute. She again expectorated some blood-tinged sputum, but her general condition was fairly satisfactory.

Fig. 9.—The gross specimen. The total weight was 3,100 Gm. or 6.8 pounds.

Twenty-four hours after operation her general condition was fair, pulse was still rapid, 116 per minute but of good quality, blood pressure 85/55, and respirations 22 per minute. The patient complained of air hunger and thirst. Hemoglobin determination revealed only 48 per cent and serum bilirubin was 5.6 mg. per cent. Pressure reading of the left pneumothorax showed a pressure of +2, –12 (centimeters of water). Despite the elevated bilirubin, 650 c.c. of blood and 500 c.c. of saline solution were given intravenously over the course of seven hours. Following this the blood pressure rose to 130/70 and patient improved definitely.

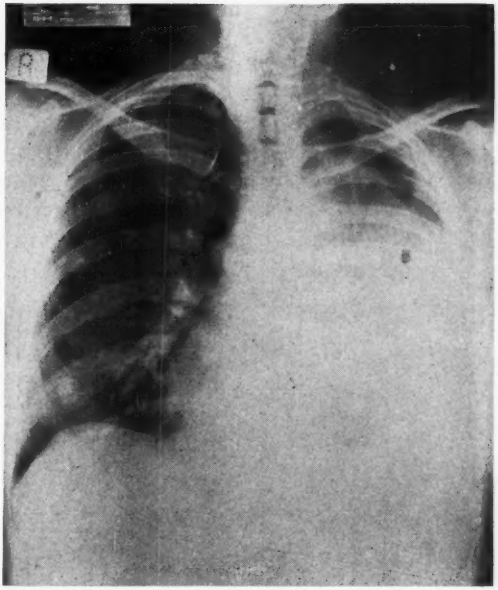


Fig. 10.—Radiograph of chest six months postoperatively. Mediastinal contents have returned to normal position. There is some contraction of left hemithorax and elevation of left diaphragm. Organization, fibrosis, and pleural thickening have taken place on the left side.

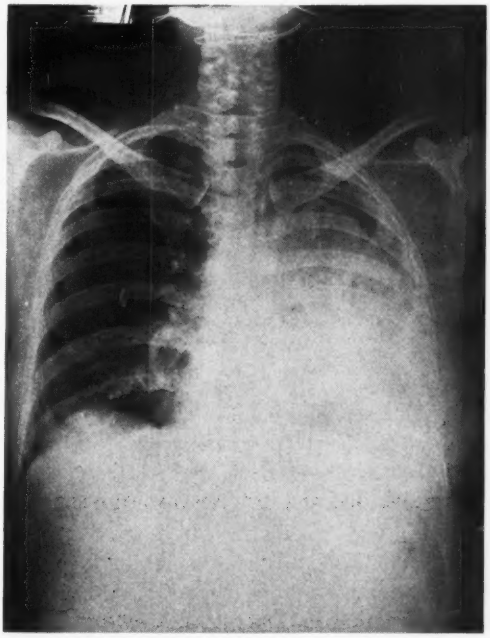


Fig. 11.—Chest radiograph one year postoperatively. There is further displacement of the mediastinal contents to the left, with no evidence of re-expansion of left lung.

Two days postoperative the blood pressure was 120/60, pulse of good quality, and respirations full and about 20 per minute. The postoperative course hereafter was quite smooth, although the patient ran a low-grade fever with temperature up to 101° F. for several weeks. On the eighth postoperative day thoracentesis revealed a pressure of +12, –3 (centimeters of water) in the left hemithorax. After 250 c.c. of air and 500 c.c. of serosanguineous fluid were aspirated, pressure was reduced to +3, –8 (centimeters of water).

Fig. 12.—The operative scar one year postoperatively.

The patient was discharged from the hospital on the twenty-fourth postoperative day and has since had an uneventful convalescence. One year after operation, she felt better than ever. She weighed 150 pounds, had no dyspnea on moderate exertion, and had no chest pain, cough, or dysphagia. She maintained that she was stronger and had more endurance than before.

A chest radiograph six months after operation (Fig. 10) showed return of the mediastinum to approximately normal position and the presence of fibrosis, pleural thickening, and some contraction of the left hemithorax. Another chest radiograph taken one year after operation

revealed further displacement of the mediastinal contents to the left side of the chest. The left lung had not re-expanded (Fig. 11). A repeat bronchospirometry six months postoperatively is of interest. It revealed marked improvement of ventilation and oxygen consumption by the right lung and slight increased ventilation but still no oxygen absorption by the left lung (Fig. 5). Apparently the left bronchial tree had opened considerably but the parenchymal portion of the lung was still atelectatic. The left lung continued to be essentially functionless. Fig. 12 shows the patient one year after operation.

CONCLUSION

A case of intrathoracic lipoma is presented. The tumor arose from the mediastinum, was entirely intrathoracic, and weighed 6.8 pounds. It was successfully excised. The operative procedure and laboratory studies are described. A short résumé of the literature on intrathoracic lipomas is included.

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DEATH FROM AIR EMBOLISM FOLLOWING BRONCHOSCOPY

Case Report

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ALTHOUGH all writers who discuss pneumothorax mention air embolism from puncture of a vessel by the needle as one of the possible serious complications, and all operators fear it and are constantly on the lookout for it, I can find no mention among the references, to which I have access, of air embolism following bronchoscopy.

It is a matter of general knowledge that septic infections of the lungs such as abscess and bronchiectasis, not uncommonly give rise to metastatic, and presumably embolic, abscess of the brain. The question of air embolism after bronchoscopy does not, however, appear to have received much attention, and in Chevalier Jackson's book *Bronchoscopy Oesophagoscopy and Gastroscopy* (1934) I can find no mention of it.

The following case report is offered as being of interest in that it deals with an apparently rather rare, but possible and dangerous, complication of a very commonly used diagnostic and therapeutic procedure.

The patient was a colored man 30 years of age. He had had, in 1935, a putrid abscess in the lower lobe of the right lung. This had previously been treated not by open operation, but by artificial pneumothorax which had been carried on for nine months before it was abandoned as useless. The patient came under my care in the latter half of 1937, while the lung was re-expanding.

Lipiodol studies revealed advanced cylindrical and saccular bronchiectasis at the site of the old abscess, and he was put on a routine of postural drainage which satisfactorily controlled the odor of the sputum, and permitted him to return to his work as a baker. From time to time, however, recurrence of odor to the sputum caused him to seek medical advice, and on these occasions I found that bronchoscopic aspiration of the secretions helped considerably for a time. Between August, 1937, and August, 1939, he was bronchoscoped and aspirated some fourteen or fifteen times, and as the relief he obtained was very considerable, lobectomy, although it had been discussed, was never very strongly urged. On Sept. 27, 1939, he came into the hospital for one of his routine bronchoscopies, prepared as usual to stay for a few hours after the procedure was over. He was bronchoscoped at 3:00 P.M. and about one-half ounce of pus was aspirated from the right side. There was a very slight trickle of blood from one of the lower bronchial branches as the aspirator was withdrawn from it, but as this had been observed

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before and there was no more than the usual resistance from the narrowed bronchus to the passage of the aspirating tube, no more was thought of it. At about 4:30 P.M. I received a telephone call to say that the patient was behaving in a peculiar manner and trying to get out of bed when told by one of the nurses to lie quiet. Psychosis of some sort was feared, and morphia was prescribed. About one-half hour later another call came to say that the patient was in convulsions. (This was about one and three-fourths hours after the bronchoscopy.) Initial convulsions had occurred in the right leg and arm, and these had quickly become generalized, causing marked spasms, with the patient in opisthotonos. On my arrival a few minutes later the convulsions were giving place to coma which lasted until his death at 5:45 P.M., i.e., slightly under two and three-fourths hours after bronchoscopy.

Cerebral embolism appeared the most likely diagnosis, and this was confirmed by ophthalmoscopic examination of the ocular fundi before death occurred. This examination showed bubbles of air streaming through all the vessels of the left retina with a few also visible in the vessels of the right eye. Examination was continued until the heart finally ceased to beat, and the air bubbles gradually came to a standstill. From the history of convulsive movements starting in the right limbs, it must be assumed that the initial cerebral lesion was a left-sided one, and this appears to be borne out by the much greater quantity of air visible in the left eye. I was informed afterward, by a doctor who was present at the bronchoscopy, that he had noticed a slight twitching of the patient's right foot as he left the table, but that he had paid no attention to it.

Autopsy revealed extensive saccular and cylindrical bronchiectasis of the basal portion of the right lower lobe. There was only a minute amount of blood in the bronchi, but at one spot there was a very small tear in the wall of a 2 mm. vein. It is possible that this tear was made at the autopsy, but as it was being specifically sought for, more than usual care with the dissection was exercised, and it is probable that the tear found was the one made by the bronchoscopic aspirator, and the one that must have admitted the air to the circulation.

The findings in this case suggest that treatment should consist of keeping the patient head down in an elevated bed on any occasion where bleeding is seen to occur after deep instrumentation of the bronchial passages, unless one can be reasonably certain that it results only from superficial trauma to the bronchial mucous membrane.

SERIOUS HEMORRHAGE DURING CLOSED INTERNAL PNEUMONOLYSIS

Case Report

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SEVERE hemorrhage during the operation of closed internal pneumonolysis is feared by all who perform this operation, but fortunately is seldom encountered. John Alexander in his book *The Collapse Therapy of Pulmonary Tuberculosis* (1937) writes as follows: "The promptness with which a thoracotomy should be performed depends upon the estimated speed with which the patient is losing blood. I know of no case in which a thoracotomy has been performed." On the same page he adds a footnote to say that such an operation had since fallen to the lot of one of his own staff, who had found it necessary to open the chest for hemorrhage from an artery that appeared to be either an intercostal or the internal mammary. In this case the adhesion was being enucleated from the chest wall by extrapleural stripping, using the electrosurgical method.

It is probable that a number of the larger adhesions, and particularly those which have been subjected to positive pressure pneumothoraces in an effort to stretch them and render them amenable to section, have by their pull upon the chest wall actually drawn into their parietal root a loop of blood vessel which would normally lie in the parietal tissues. (See Fig. 1.) A glance at the diagram will serve to make this suggestion clear. In such a case, section of the adhesion as near the chest wall as possible—the usual method—may open up a blood vessel of formidable proportions, and render immediate thoracotomy an absolute necessity.

Hemorrhage is the gravest emergency which the surgeon is called upon to face, and in its presence, with a life in the balance and only a few minutes in hand, the use of any method which will control it is more than justified. The case reported here occurred in December, 1937, in a small hospital in Kingston, Jamaica, and the vessel opened, although I was never able to prove it, was in all probability the subclavian vein. The result of such a catastrophe can better be imagined

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than described, and this report is offered with the idea of urging the need for all possible speed in dealing with such an emergency.

The patient, a colored male, 22 years of age, had had pneumothorax induced on the left side for an extensive upper lobe tuberculous lesion in the center of which lay a cavity slightly more than 5 cm. in diameter. Two groups of adhesions, lateral and apical, rendered collapse unsatisfactory, and on Nov. 25, 1937, the lateral group was divided, while a partial section of the apical mass was also done. Just over two weeks later, on Dec. 13, 1937, an attempt to complete the separation of the apical residue was made. The instrument used was that of Jacobaeus, and the adhesion was being cut through as close to its parietal attachment as was possible. All went well for about one half of the way,

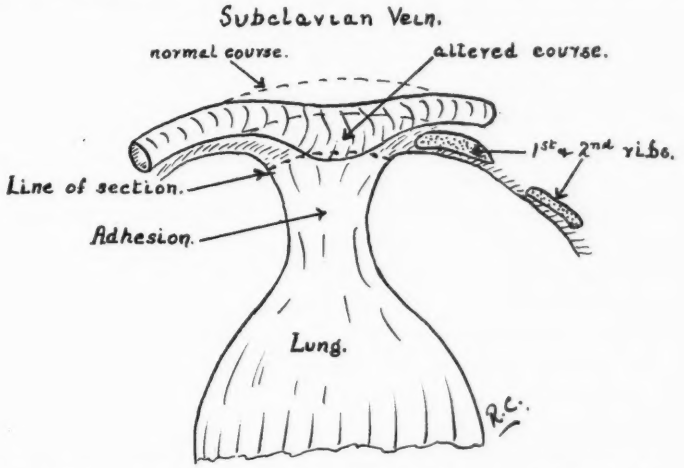

Fig. 1.—Diagram to illustrate possible bulging of subclavian vein into base of large adhesion as a result of traction on the wall of the vein. Attempted section, as shown by lowest dotted line, may open the vein.

when suddenly the point of the cautery seemed to sink into the tissues, and there appeared an enormous gush of dark blood, which almost instantly obscured the lens of the thoracoscope. Control of such a hemorrhage with the cautery point was obviously impossible, and time would only have been wasted in an attempt.

Immediate open thoracotomy was the only alternative. No instruments for the purpose had been sterilized, and there was no one available who could be called upon to give an immediate anesthetic. The magnitude of the hemorrhage was such that not a second was to be lost, if the patient's life was to be saved, and it was decided to open the chest without any form of anesthesia, as even a local infiltration would undoubtedly have taken so long that death would have occurred before it

was finished. The patient was turned onto his back and iodine was literally spilled all over his chest. The scalpel, which fortunately was sterile, was entered in the fourth interspace in the midaxillary line, and with one sweep was carried round along the interspace to within one inch of the sternum, dividing skin, fascia, and pectoral muscles at once. A second stroke divided intercostal muscles and pleura, and opened the thoracic cavity widely. Blood loss had been so rapid that the patient was almost unconscious, and only groaned slightly while subjected to this prehistoric form of surgery.

Forcible retraction was made by the assisting nurse with one hand, while the operator's left hand was introduced into the thoracic cavity in an attempt to locate and stop the bleeding vessel with a finger. It was intended to plug the whole apex of the chest with gauze, but there were only a few small swabs available, and the use of surgical dressing towels had to be resorted to. Two of these were opened out, and one by one were introduced into the chest, while the left hand, still in position, packed them firmly up into the apex. The urgent need was to stop the hemorrhage by any method available, and the lung had to be left to take care of itself as best it might. The time from the moment the hemorrhage started until the pack was in place was just under five minutes. When the towels had been inserted the end of the lower one was left protruding through the wound, which was covered with another, being allowed to remain unsutured, and the patient was returned to bed and treated for hemorrhage. No blood for transfusion was available, and glucose and saline alone could be used. The patient was almost pulseless, and his mucous membranes appeared to be practically drained of blood. By that night, however, he had made a good enough recovery to permit the removal of the rather coarse towels and their replacement with a roll of much softer gauze. There was still some oozing in the vicinity of the initial vessel wound, but this appeared to be well controlled by the gauze. Blood in the chest was aspirated and clots were removed from the lower half of the chest; together they represented about 1000 to 1200 c.c. of shed blood, and the thoracotomy wound was sutured with through-and-through stitches, leaving the gauze in place. Two days later the wound was reopened and the gauze pack was removed. There was now no oozing at all, and the chest was firmly closed layer by layer. The patient ran a considerable temperature, and when the stitches were removed there was infection in the lateral half of the wound. At the same time aspiration of the chest yielded pus from which no tubercle bacilli could be obtained, but which contained staphylococci and streptococci. This empyema was drained and eventually cleared up, leaving a soundly healed chest wall and a re-expanded lung, with unclosed cavity.

The patient was advised to have a left thoracoplasty, but spread of disease on the right side occurred. He was admitted to this hospital on its opening, and pneumothorax on the right was induced in an attempt to control the spread on that side, in order to prepare the way for a left thoracoplasty. It was not successful in accomplishing this, however, and he returned to his home where he died in 1942, nearly five years after the event reported.

The surgery that was used here is admittedly what I have referred to as prehistoric, but it was the only kind available, and in this case proved lifesaving.

THORACOPLASTY FOR TUBERCULOSIS

Late Results

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IN THIS article is presented a review of 232 cases of thoracoplasty for pulmonary tuberculosis performed at Koch Hospital, Koch, Mo., during the ten-year period from 1931 to 1941. Koch Hospital is the St. Louis municipal tuberculosis sanatorium for both white and colored patients, male and female, and has a capacity of 700 beds.

The follow-up (100 per cent) was obtained through the tuberculosis section of the hospital division of the St. Louis Public Health Service records of which are kept up to date by the outpatient department and the visiting nurses.

The patients are chosen for surgery after a period of treatment and observation varying from six months to two years. The surgery on all patients at Koch Hospital is done by the resident in surgery and has lately been under the general supervision of Dr. Evarts A. Graham, head of the department of surgery at Washington University School of Medicine, and Dr. J. L. Mudd of the staff of St. Louis University School of Medicine.

Before a decision is made on any patient at Koch Hospital each case is thoroughly discussed at a biweekly staff meeting, medicosurgical, where the patient's history is reviewed and his status considered in the light of clinical, roentgenologic, bacteriologic, and hematologic studies and recommendation for treatment made.

Table I

	Male	Female	White	Colored	Av. Age (Yr.)
Group I	27	54	65	16	31.5
Group II	31	48	46	31	32.0
Group III	37	37	47	27	31.3
Totals	95	137	158	74	

Group I consists of those patients operated upon between the years 1931 and 1935; Group II, between 1936 and 1938; and Group III, between 1939 and 1941 inclusive.

Thoracoplasty was indicated in cases where the pneumothorax was unsuccessful or was successful but ineffective. Pneumothorax was tried and found ineffective in 90.5 per cent of the cases operated upon. Phrenic crush was done unilaterally on 80 patients and bilaterally on 2 patients.

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Every patient had positive sputum prior to operation and except in two cases all had visible cavitation, 19 having had bilateral cavitation. Thirteen of the patients also had either mixed or tuberculous empyemas and two had bronchiectasis.

In some cases thoracoplasty was performed on patients who had stabilized contralateral lesions and tuberculous and mixed empyemas, with fair results; however, a contralateral progressive lesion, extrapulmonary foci, cardiovascular insufficiency, and serious degenerative diseases were considered contraindications to thoracoplasty. Patients with additional tracheobronchial disease were first treated bronchoscopically and then by thoracoplasty; in this way better results were obtained.

It has been the policy of the Koch Hospital staff in cases with bilateral cavities to treat one side by other collapsing measures (pneumothorax, pneumonolysis, phrenic crush, or pneumoperitoneum) and then when the lesion on that side is under control, recommend thoracoplasty for the opposite side.

We present the cases divided into three groups: Group I, cases operated upon from 1931 to 1935 inclusive; Group II, cases from 1936 to 1938 inclusive; Group III, cases from 1939 to 1941 inclusive, because the selection of the earlier cases for thoracoplasty was made according to different criteria, and the surgical procedure was often not sufficiently extensive. These two factors make it impossible to get a clear picture of the results without separation into three periods. In the group of 232 thoracoplasties there were 125 performed on the right side, 105 on the left side, and 2 bilateral.

In Groups I and II the technique used was different from that used in the operations in Group III. In Groups I and II an average of five ribs was removed in each stage, two stages being done. In Group III there was removed an average of three ribs at each stage, beginning with the removal of the upper ribs and allowing three weeks' time to elapse between each stage. Also in Group III, in poor-risk patients only two ribs were removed at each stage.

TABLE II

	1 STAGE	2 STAGES	3 STAGES	4 STAGES	5 STAGES	6 STAGES
Group I	16	50	8	3	2	1
Group II	17	44	14	1	2	
Group III	5	39	27	3		

This table shows the number of stages of thoracoplasty performed in each of the three groups.

From Table III it is interesting to note the fewer revisions necessary in the cases done in the last two periods, which evidences the great improvement in the technique used in the later two groups over that used in the first group. In the second and third groups, each rib was removed along with its transverse process.

TABLE III

	NUMBER OF REVISIONS
Group I	42
Group II	6
Group III	2

This table shows the number of revisions performed in each group.

The average hospital stay for the patients subjected to thoracoplasty in Group I was 4.7 years, in Group II, 3 years, and in Group III, 2.3 years. It is worthy of note that the hospital stay has been progressively shortened during the past ten years until in Group III it is one-half of what it was in Group I. This is probably due to a better selection of cases, better operative technique, and better preoperative and postoperative care.

TABLE IV

	NO. OF CASES	ARRESTED		QUIESCENT		IMPROVED		UNIMPROVED		DEAD	
		NO.	%	NO.	%	NO.	%	NO.	%	NO.	%
Group I	81	26	32.8	12	14.8	9	11.1	5	6.1	29	35.8
Group II	77	33	42.8	16	20.8	8	10.4	1	1.3	19	24.5
Group III	74	39	52.7	16	21.6	4	5.4			15	20.2
Totals	232	98		44		21		6		63	

This table summarizes the end results of the 232 cases of thoracoplasty discussed in this communication.

In Table IV we have shown the results divided into the number of patients with arrested condition, number quiescent, number improved, number unimproved, and the number dead. However, considering those cases charted as arrested, quiescent, and improved as being benefited by thoracoplasty, Group I shows favorable results in 49.7 per cent of the cases, Group II in 74.0 per cent of the cases, and Group III in 78.7 per cent of the cases.

In Group I there was only one spread to the contralateral lung and in Group II only one, but there were five cases of contralateral spread in Group III.

TABLE V

	OPERATIVE	2 YR. AFTER OP.	4 YR. AFTER OP.	6 YR. AFTER OP.
Group I	3	11	11	4
Group II	2	10	5	1
Group III	6	7	2	

Analysis of deaths occurring in the three groups of cases.

Table V gives an analysis of the deaths, divided into operative and nonoperative, occurring in the three periods. In order to show the length of survival of the nonoperative deaths, we have divided them into those cases in which death occurred within two, four, and six years of operation. Death in each case charted was due to tuberculosis; however, in this series there were five other deaths due to nontuberculous causes.

SUMMARY

The end results of 232 consecutive thoracoplasties for tuberculosis, done at Koch Hospital, are presented. The results are based upon 100 per cent follow-up to date on all cases.

The cases are divided into three groups because the series extends over a period of ten years, and during this time there have been changes in the indications for thoracoplasty and in the operative technique. In Group I, 58.7 per cent of the patients were improved by thoracoplasty; in Group II, 74.0 per cent were improved; and in Group III, 79.7 per cent were improved. The total number of both early and late deaths has decreased from 35.81 per cent in Group I to 20.2 per cent in Group III. Likewise, the hospital stay has been reduced to one-half of what it was at the beginning of the series.

PSEUDOBRONCHIECTASIS*

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AN ACUTE respiratory infection associated with pulmonary lesions has received considerable attention during the past few years. Interest has been stimulated further because it has reached epidemic proportions sufficient to incapacitate temporarily a large number of men in the Armed Forces. The disease has been variously designated as bronchopneumonia of unusual and undetermined etiology; atypical pneumonia with leucopenia; pneumonitis; acute influenzal pneumonia; and acute bronchiolitis with associated atelectasis. Common usage, however, has parcelled these and various other names into a loose term, atypical pneumonia. Failure to find a pathogenic organism and some of the clinical features of the disease has made virus pneumonia another attractive possibility.

The condition is usually self-limited and has few complications. Fortunately the mortality rate is low. The common surgical complication of bacterial pneumonia, namely, empyema, is rare. Certain late features of the disease, however, are of interest to the thoracic surgeon. This communication will emphasize the importance of the effects of the disease on the bronchi and the production of pseudobronchiectasis.

Roentgenographic Manifestations.—The peculiar x-ray shadows in the lungs associated with primary atypical pneumonia have been described by various investigators. The roentgenograms in these cases are usually characterized by mottled densities; and lobular and lobar atelectasis are common with wide variance of distribution and extent. Sometimes the roentgenographic manifestations of the disease are bizarre and misleading. For example, differentiation between atypical pneumonia and pulmonary tuberculosis may be impossible if only one x-ray film of the chest is available and can only be made after comparison of several roentgenograms and examination of the sputum for tubercle bacilli. In fact, Kneeland and Smetana[1] have reported a case of atypical pneumonia in which the appearance of the x-ray film was so typical of tuberculosis that pneumothorax was instituted before the true nature of the condition was established. Not only tuberculosis but other potentially surgical diseases of the chest may be imitated, both clinically and roentgenographically. A sharply localized zone of atelectasis may strongly sug-

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gest a pulmonary tumor, and in many instances basilar densities in the roentgenogram of a patient with a chronic productive cough might easily lead to a preliminary diagnosis of bronchiectasis.

PSEUDOBRONCHIECTASIS CAUSED BY ATYPICAL PNEUMONIA

Delineation of the bronchi with radiopaque oil will, under ordinary circumstances, settle the diagnostic problem when bronchiectasis is suspected. We have been able to demonstrate, however, that following atypical pneumonia lipiodol bronchograms may occasionally be misleading and give the erroneous impression that the condition is true bronchiectasis. The temporary nature of the bronchial dilatations has been proved by visualization of the bronchi a few weeks later showing a complete return to normal of the bronchial configuration. Speculation on the possibility that in some instances permanent damage to the bronchi may result from the effects of atypical pneumonia would be premature. Atelectasis is a prominent feature of the disease and may persist for many weeks. Campbell and associates,[2] in their excellent paper on atypical pneumonia, describe a case in which the right lower lobe remained partially atelectatic for a period of forty-six days. A bronchogram demonstrated not only atelectasis but also localized fusiform bronchiectasis. Our experiences suggest that this may be another example of "false bronchiectasis" and that it is possible that subsequent visualization might reveal a normal bronchial tree. The final consequences of prolonged atelectasis and infection associated with some cases of atypical pneumonia can be evaluated only when complete data are available. It seems possible, however, that the damage to the bronchi in severe cases could result in irreversible changes.

Experienced students of thoracic disease are aware that the bronchi of atelectatic or partially atelectatic lobes may seem short and wide when filled with radiopaque substance. When the effect is marked, bronchograms may give the impression that the condition is bronchiectasis. The residual bronchial dilatations which sometimes complicate atypical pneumonia are probably not produced entirely by this mechanism since they may be present when evidence of concomitant atelectasis is lacking, and moreover the dilatations may often exceed the cylindrical configuration generally produced by temporary atelectasis.

Ochsner,[3] in a study of patients suffering from chronic coughs, found that 90 per cent of the group had demonstrable bronchial dilatations. In four cases the bronchial enlargement disappeared. Altmann and Engel,[4] in 1933, demonstrated reversible cylindrical dilatations in the bronchi of children following acute upper respiratory infections. Fleischner,[5] in his important article "Reversible Bronchiectasis," describes a case in which grossly dilated bronchi were demonstrated. Seven months later the bronchi had resumed normal configuration. Delinea-

tion of the bronchi at a later date, however, again revealed bronchiectasis and a lobectomy was performed.

Fleischner condemns the term pseudobronchiectasis because he considers reversible bronchiectasis can represent an early stage of true bronchiectasis. We are skeptical of this conception of the disease and have elected to employ the term pseudobronchiectasis for our cases.

One of four representative cases which we have studied is presented to illustrate the diagnostic problem.

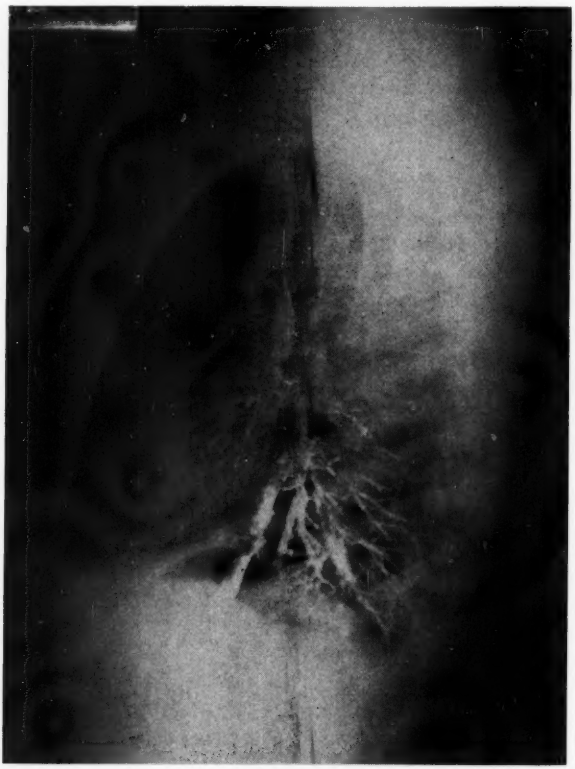

Fig. 1A.—Bronchogram demonstrating bronchiectasis of left lower lobe.

Case Report.—A 24-year-old officer was transferred to Walter Reed General Hospital with the diagnosis of bronchiectasis of the left lower lobe. His chief complaint on admission was a cough productive of thick purulent sputum. The onset of the acute illness six weeks before had been characterized by fever ranging from 100 to 102° F., pain in the left chest, and malaise. Roentgenograms of the chest during the first week of the disease revealed a zone of opacity in the left lower lobe. The original diagnosis was atypical virus pneumonia. Sulfonamide ther-

apy was not effectual, but the acute symptoms subsided in about one week and the only residual was a chronic productive cough. Examination of the blood, urine, and smears and cultures of the sputum did not contribute to the diagnosis.

A lipiodol bronchogram made two weeks after subsidence of symptoms revealed what was considered to be extensive bronchiectasis of the left lower lobe. At that time there was no evidence of atelectasis on the conventional roentgenogram. Delineation of the remainder of the left

Fig. 1*B*.—Appearance of bronchi in the same case two months later showing complete return to normal.

lung and of the right lung proved that the bronchial dilatations were limited to the left lower lobe. Accordingly, the advisability of surgical intervention was considered and the patient was transferred to the thoracic surgical section. The decision to delay the operation was caused chiefly by the complete lack of a history of pulmonary disease preceding the acute illness and by the fact that the sputum was not foul in character. The material was thick, purulent, and tenacious, but the characteristic putrid odor of bronchiectasis was lacking. It is note-

worthy, however, that the patient himself considered the sputum to have an unpleasant, foul odor.

During a period of observation for two weeks, improvement was slow and the productive cough continued. The officer was given sick leave and two months after the original bronchograms showing bronchiectasis were made, the left lung was again visualized with radiopaque oil. The bronchial dilatations had completely disappeared (Figs. 1*A*, and 1*B*). All symptoms gradually subsided. The officer returned to duty and has been well for more than a year.

Fig. 2*A*.—Original bronchogram which revealed bronchiectasis of left lower lobe and lingula of the left upper lobe.

Five other cases with almost identical histories have been observed. Bronchograms, following an acute illness diagnosed as atypical pneumonia, have demonstrated bronchial dilatations but after a period of one or two months the configuration of the bronchi have returned to normal.

Figs. 2*A* and 2*B* demonstrate the changes in the bronchial tree in another case. The original bronchogram had the appearance of bron-

chiectasis. The second bronchogram shows only two or three bronchi that still seem enlarged. During this interval the productive cough had diminished in severity. The history of the disease is, for practical purposes, identical with Case 1.

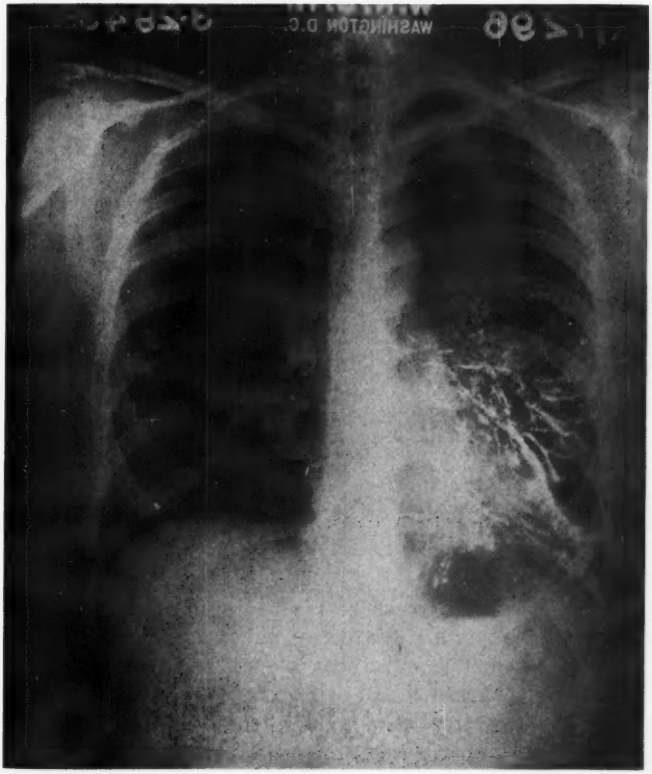

Fig. 2*B*.—The same case one month after the original bronchogram, most of the bronchi have resumed a normal configuration.

DIFFERENTIAL DIAGNOSIS

The clinical features of atypical pneumonia have received considerable attention and are adequately described in numerous articles on the subject. Apparently, the diagnosis is made by exclusion of other pulmonary diseases, by failure of a pneumonia-like lesion to respond to sulfonamide therapy, and by the roentgen appearance of the lungs.

Although the symptoms and signs vary widely, the disease is often characterized by a gradual onset, and the earliest symptom is usually a dry cough which later becomes productive and often paroxysmal. Chilly sensations and sometimes chills and fever become evident as the infection progresses. Unfortunately, all of these manifestations are com-

patible with practically any variety of pulmonary disease and will not, therefore, be reliable in differentiating the precise nature of the lesion. A more abrupt onset, and in a few instances hemoptysis, may further increase the diagnostic dilemma. The physical findings vary considerably and are notoriously unreliable in establishing a positive diagnosis. If one adds to the clinical evidence a bronchogram demonstrating what seems to be definite anatomic bronchiectasis, the logical assumption is apt to be that the acute phase of the disease is secondary to pre-existing bronchiectasis. In the case just described the patient narrowly escaped unnecessary lobectomy under similar circumstances.

Our experiences suggest, however, that a critical analysis of the history, careful examination of the sputum, and the appearance of the bronchial tree when examined with the bronchoscope will warn the thoracic surgeon that the disease may not be true bronchiectasis, provided that he is aware that atypical pneumonia can produce temporary dilatations of the bronchi.

CLINICAL HISTORY

The first clinical point which deserves careful attention is the history of chronic cough before the onset of the acute illness. It is important to emphasize that an affirmative answer to the question of whether a cough is chronic requires careful consideration since practically all individuals cough occasionally and unless the character of the cough has changed or has become productive the symptom may be of questionable significance.

In advanced cases of bronchiectasis the productive cough, clubbed fingers, and other stigmas of the disease will make the diagnosis obvious. Difficulties in differentiating between the residual pseudobronchiectasis of acute atypical pneumonia and true bronchiectasis will occur in early lesions and in instances of so-called dry bronchiectasis where the bronchial dilatations have not become infected and the symptoms are minimal. Even in these cases, however, careful questioning will usually establish the existence of a troublesome cough before the onset of the acute illness.

SPUTUM EXAMINATION

Little factual evidence can be expected from the microscopic examination of the sputum. It is true that careful anaerobic cultures of the material coughed up in advanced bronchiectasis cases will often demonstrate *Spirilla,* fusiform bacilli, Bacterium melaninogenicum, and other anaerobic organisms which are uncommon in the sputum of atypical pneumonia. When the condition is this far advanced, the characteristic putrid odor which is always associated with the anaerobic organisms makes the laboratory evidence of academic interest only. A great variety of aerobic organisms will be found in the sputum of patients with either condition and unless practically pure cultures of

a single organism are found on repeated examinations the aerobes are of no diagnostic significance. However, a study of a twenty-four-hour sample of the sputum is valuable. Characteristically, the sputum of bronchiectasis when allowed to stand will separate into three layers: The upper layer is colorless or slightly greenish and contains air bubbles, pus, and mucus. The second layer is similar to the first but has less air. The third layer consists of a greenish sediment which is thick and is made up of pus cells, fat rests, Dittrich's plugs, and detritus. The sputum of pseudobronchiectasis may be copious, is often frankly purulent, sticky, and tenacious, but does not have a tendency to separate into layers on standing. Blood streaking and occasionally frank hemoptysis may be associated with either disease, but is more common in bronchiectasis. The total amount of sputum is variable from day to day in both conditions.

BRONCHOSCOPIC EXAMINATION

Probably more important information can be obtained by inspection of the bronchial orifices and bronchial mucous membrane through the bronchoscope than by any other diagnostic procedure. In cases of bronchiectasis, even in a dry phase, the material obtained by inserting a suction tip into the involved lobe will usually have a fetid odor. The bronchus or bronchi to the diseased lobes will be edematous and reddened. Pus will flow from the orifice of the bronchus and large quantities of purulent secretion may be forced out by respiratory movements or coughing. The bronchial mucous membrane on the affected side, and particularly about the lumen of the bronchi, is reddened but not strikingly edematous. It is easy to localize precisely the origin of the pus in terms of bronchopulmonary segments and the adjacent mucous membrane will show only the effects of being bathed in the purulent secretions coming from the infected lobes. The tissue bleeds easily and aspiration of the diseased lobes with a suction tip may provoke rather brisk bleeding. After careful removal of the accumulated secretions, temporary improvement may be expected in many bronchiectasis cases.

The appearance of the bronchial tree in pseudobronchiectasis after atypical pneumonia is usually quite different. Pus can be seen coming from the bronchi of the involved lobe or segments, but usually the entire surface of the visible mucous membrane is markedly edematous. A generalized inflammatory reaction is evident, which from its gross appearance might be described as "suppurative bronchitis." The tissues are not as friable as in the bronchiectasis cases and manipulation with the instruments rarely results in troublesome bleeding. Even when an enormous quantity of pus is obtained by suction, the characteristic odor associated with bronchiectasis is not evident. Although variations will occur with each case, the temporary alleviation of symptoms common after bronchoscopic aspiration in bronchiectasis cases is usually not so evident or is entirely absent in patients with pseudobronchiectasis.

DISCUSSION

It must be emphasized that the information which is available from the history, examinations of the sputum, and inspection of the bronchi with a bronchoscope will only suggest that bronchial dilations evident on a bronchogram may not represent true anatomic bronchiectasis. A positive diagnosis of pseudobronchiectasis is only justifiable when subsequent visualization of the bronchial tree reveals normal configuration of bronchi which were formerly dilated.

Categorical statements on the length of time pseudobronchiectasis resulting from atypical pneumonia may persist are impossible. Probably in a few cases the bronchi will remain enlarged for as long as three months. In most instances, however, at least part of the bronchi will have returned to normal in four to six weeks. There is, of course, a distinct and interesting possibility that when severe injury has occurred, particularly if the muscle and cartilage are involved, the damage may be permanent. Further experience will be necessary before this question can be answered.

If one is aware that the rather ill-defined disease of atypical pneumonia is capable of producing temporary dilations of the bronchi, it would be easy to consider for a time an early or dry bronchiectasis case to be an example of pseudobronchiectasis. This would not jeopardize the patient's future health or constitute a grave error in clinical judgment; moreover, progression of symptoms and repeated demonstration of the bronchial dilations would soon establish the correct diagnosis. The test of time should be infallible and scrupulously followed when any doubt exists as to the true nature of the condition. Bronchiectasis is a chronic progressive disease, the manifestations of which will soon become unmistakable. Failure to consider the possibility of pseudobronchiectasis might, however, result in unnecessary resection of lung tissue. It is important, therefore, to record that atypical pneumonia can produce temporary enlargement of the bronchi and to urge that surgical intervention be delayed in questionable cases until repeated bronchograms and the clinical features of the disease establish an unequivocal diagnosis of anatomic bronchiectasis.

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THRESHOLD VARIATIONS TO VAGUS NERVE STIMULATION

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IT IS commonly believed that irritation of the vagus nerve during an intrathoracic operation is likely to result in circulatory reactions. Capps and Lewis[1] focused attention on vagal reflexes during thoracic manipulations in 1908. In their experiments the stimulation was applied to the pleura and the circulatory response has since been designated by the term they used, namely pleural reflexes. Recently it was shown that in the dog[2, 3] and in man[4] stimulation of the abdominal vagus produced little or no circulatory response. Such observations prompted an investigation to determine whether differences in sensitivity of the vagus trunk at different levels could be demonstrated more thoroughly.

Two hundred and two observations were made on nine dogs and four cats. The animals were anesthetized by the intravenous injection of chloralosane, 90 mg. per kilogram, or nembutal, 30 mg. per kilogram. A tracheal cannula was then inserted and connected to a Starling motor pump which induced mechanical respiratory movements at the rate of 24 per minute. The vagi were isolated (a) in the neck, (b) in the chest above the heart, (c) in the chest just above the diaphragm, and (d) in the abdomen just above the celiac plexus. The arterial blood pressure, obtained by cannulating the left femoral artery in dogs and the left common carotid in cats, was recorded with the aid of a mercury manometer and a moving drum. A Harvard induction coil with two fresh dry cells was the source of faradic current for stimulation. The strength of stimulus chosen, which was of the same intensity and for the same length of time in each instance, was that which produced a minimal fall in blood pressure when applied to the cervical portion of the vagus.

RESULTS

Results were invariably constant. As represented in Fig. 1, the application of a minimal stimulus upon either intact cervical vagus produced a small response characterized by bradycardia and a fall in mean arterial blood pressure of 15 per cent. The same stimulus upon the same vagus nerve in the chest above the heart resulted in temporary cardiac asystole and a fall in arterial blood pressure of 80 per cent. No circulatory changes were observed when this same stimulus was applied

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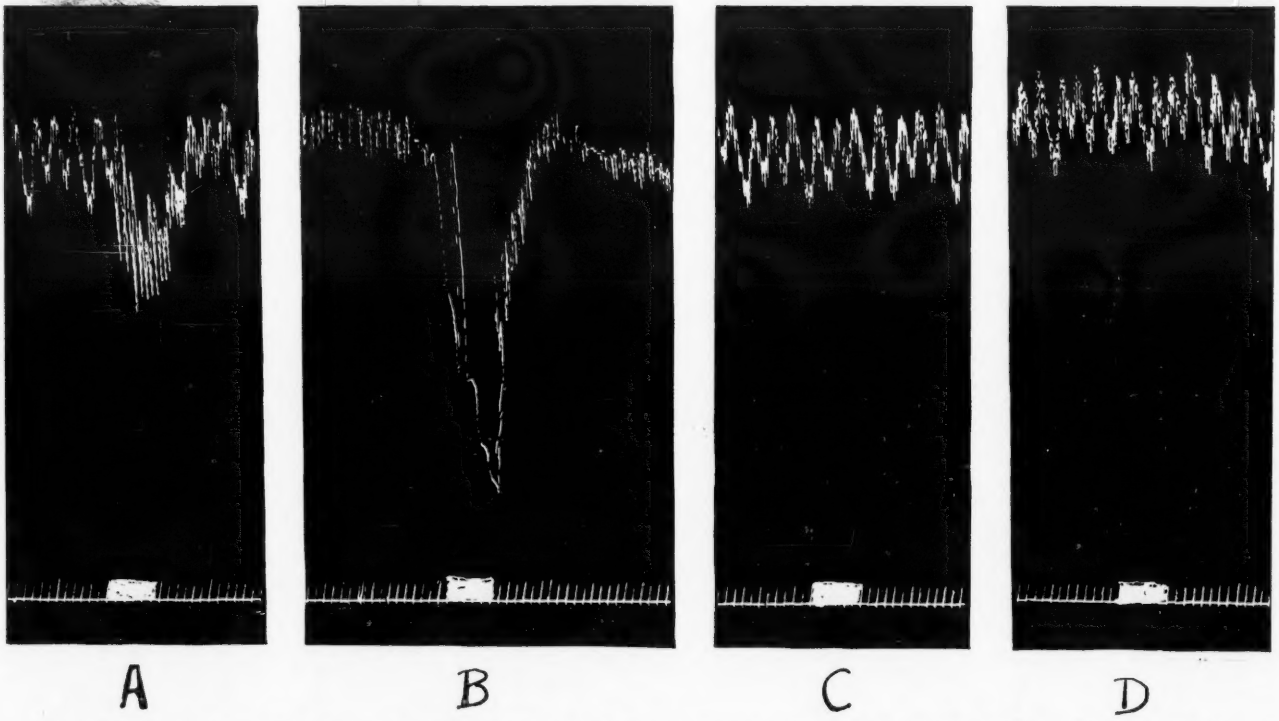

Fig. 1.—Dog of 14 kg. Chloralsane anesthesia, 90 mg. per kilogram; control arterial blood pressure 146 mm. Hg. *A*. Application of faradic stimulus to cervical segment of left vagus. *B*. Application of same stimulus to upper thoracic segment of left vagus. *C*. Application of same stimulus to lower thoracic segment of left vagus. *D*. Application of same stimulus to abdominal segment of left vagus.

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to the same vagus trunk in the chest just above the diaphragm or to the same vagus trunk in the abdomen.

Stimuli to the abdominal vagus, causing a minimal fall in blood pressure, produced immediate cardiac asystole with a precipitous fall in blood pressure and death, when applied to the upper thoracic segment of the vagus of the same side.

To rule out the possibility of any significant involvement of the medulla and the contralateral vagus, the vagus of the opposite side was sectioned in the cervical region after the usual responses were observed. Reapplication of the same stimulus to the four different regions resulted in a response similar to that prior to section of the nerve.

DISCUSSION

The data appear to indicate that a difference in the threshold of sensitivity exists in the vagus nerve depending upon the level of stimulation. They substantiate the clinical impressions that circulatory depressant reactions occur more commonly during intrathoracic than during upper abdominal operations involving vagal manipulation.

The disparity in circulatory reactions in different portions of the same nervous tract and especially the diminution of reflex activity both above and below the upper thoracic segment of the vagus is not readily interpreted. However, several considerations may be entertained. (1) Variations in the thickness of the vagal sheath. This anatomic factor was eliminated by stimulating different parts of the denuded vagus nerve and noting that the responses were similar to those noted prior to denudation. (2) Proximity of stimulus to postganglionic synapse in the heart. The great intersynaptic velocity of the nervous impulse and the small variation in distances preclude this factor. (3) The number of fibers stimulated. While the fibers decrease in number as the nerve extends peripherally, this possibility does not explain fully why the most marked results were noted upon supracardiac thoracic (and not cervical) stimulation of the vagus. (4) Disproportion of afferent to efferent vagal fibers. There are proportionately less afferent than efferent fibers in the vagus as it continues peripherally. This may account for the results obtained upon stimulating the abdominal vagal but not the supracardiac branches. (5) Aortic depressor fibers. These supracardiac afferent vagal branches may explain the absence of effect upon stimulation of infracardiac and abdominal branches of the vagus and possibly the predominant effect of supracardiac stimulation. (6) Cardiac plexus. The vagus traverses through the cardiac plexus to its terminations and comes in close proximity with sympathetic afferent branches. It is quite possible that supracardiac stimulation of the vagus produces the marked circulatory depression noted in the data not only as a result of the afferent (aortic-depressor) and efferent (cardiac) vagal fibers, but also via sympathetic afferent pathways.

SUMMARY

Faradic stimulation of the vagus nerve at different points along its course results in varying degrees of cardiovascular depression. Using a constant stimulus, the most marked changes were noted upon stimulating the upper thoracic segment of the vagus. Lesser effects were seen when the cervical vagus was stimulated. Little or no effect followed stimulation of the lower thoracic and upper abdominal branches of the vagus.

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CHYLOTHORAX

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CHYLOTHORAX may be defined as the presence of a milky, fat-containing fluid in the pleural space. The condition occurs when chyle escapes from the thoracic duct or its radicles. Chylothorax may be the result of trauma to the duct or obstruction of the duct or of the great veins into which it empties. In 1633, Bartolet[1] first described this condition. In 1685, Diemerbroeck[2] discovered chylothorax due to rupture of the thoracic duct of a man who had died from severe trauma. About sixty-six cases of chylothorax due to trauma have been recorded. In 1935, Yater[3] estimated that eighty cases of chylothorax of non-traumatic origin had been reported. In the records of the Mayo Clinic we have found data on nine cases of chylothorax. It is of great interest to us that seven of the nine patients were seen at the clinic in the period from December, 1942, to November, 1943, inclusive. We believe that chylothorax is much more common than our records or the literature on the subject would indicate. It is our purpose to promote interest in the recognition of this condition. Because chylothorax may often be the result of trauma, it should be looked for as a complication of war injuries.

EMBRYOLOGY AND ANATOMY OF THE THORACIC DUCT

It is worth while to review briefly the embryology and anatomy of the thoracic duct.[4] Lymphatics arise independent of blood vessels from discrete mesenchymal spaces which become lined by endothelium. By progressive fusion and budding of such local anlagen the lymph system grows to its final form. Lymph sacs develop with openings into veins. The openings of the jugular lymph sac persist and are exits used by the thoracic duct and the right lymphatic duct. The thoracic duct arises in the abdomen from the cisterna chyli, a lymph sac lying over the body of the second lumbar vertebra. The cisterna chyli receives two lumbar lymphatic trunks and the intestinal lymphatic trunk. The embryonic thoracic ducts are bilateral and symmetric and have numerous cross anastomoses. The adult duct is produced by disappearance of varying portions of the embryonic ducts and growth and persistence of the remaining portions.

The anatomy of the adult thoracic duct is subject to considerable variation because of the complexity of its embryologic development. The subject has been studied carefully by Davis,[5] who described nine types

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varying from the complete persistence of bilaterally symmetric ducts to the persistence of one duct only. In 64 per cent of all subjects studied by Davis the thoracic duct was formed by the caudal portion of the right thoracic duct and the cephalic portion of the left duct. These segments are joined by a persisting cross-anastomosing branch. This type is described by all anatomists and is considered as normal. From its origin in the cisterna chyli the thoracic duct courses to the right and behind the aorta and enters the thorax through the aortic hiatus of the diaphragm. It ascends through the posterior mediastinal cavity between the aorta and the azygos vein. Opposite the fifth thoracic vertebra it inclines toward the left side and ascends behind the thoracic part of the left subclavian artery and between the left side of the esophagus and the left pleura to the upper orifice of the thorax. Passing into the neck, it forms an arch which rises about 3 or 4 cm. above the clavicle and crosses anterior to the subclavian artery. It ends by opening into the angle of junction of the left internal jugular vein with the left subclavian vein. The duct is generally flexous and constricted at intervals, presenting a varicose appearance. In the neck it is joined by the left jugular and left subclavian trunks and the left bronchomediastinal trunk.

The right lymphatic duct may be only 1 cm. long. It receives the right jugular and right subclavian trunks as well as the right bronchomediastinal trunk, which carries lymph from the right lung, right side of the heart, and part of the convex surface of the liver. The thoracic duct is 4 to 6 mm. wide and is equipped with several valves, having a pair at its termination arranged to prevent the reflux of venous blood.

MECHANISM OF THE FLOW OF LYMPH

The thoracic duct is the main collecting trunk of the lymphatic system. Flow of lymph is dependent upon several factors. Pressure in the venous angle receiving the thoracic duct may be zero, or less than atmospheric pressure. Movement of lymph from the duct is the result of slight elevation of pressure in the duct as compared with that of the subclavian vein. Movements of the extremities and intestinal peristalsis squeeze the lymph toward the thoracic duct. Respiratory movements with fluctuation of thoracic pressure facilitate movement of lymph. Lymph has been observed to continue flowing in the thoracic duct as long as four hours after death. The amount of chyle flowing through the duct varies between 130 and 195 c.c. per hour.[6]

COMPOSITION OF CHYLE

The lymph of the thoracic duct is high in protein content when hepatic production of lymph exceeds that of the intestine. It contains varying amounts of emulsified fat and during active absorption of fat lymph has a milky appearance.

There has been considerable discussion as to the physical characteristics of chylous fluid. Frequent attempts have been made to distinguish true chyle from "pseudochylous" fluids. However, it is now generally agreed that if emulsified fat can be demonstrated in pleural fluid, chylothorax is present. The character of chylous fluid will vary with such factors as the fat and protein content of the diet and the degree of dilution by ordinary pleural fluid. Hydrothorax frequently occurs in association with chylothorax. Obviously, the amounts of fat and protein in the fluid, the specific gravity, the cell count, and even the appearance of the fluid will vary to a considerable extent. The chief characteristics of chyle as listed by Brescia[7] are given in Table I.

TABLE I

CHIEF CHARACTERISTICS OF CHYLOUS FLUIDS*

1. Milky appearance—finely emulsified fat
2. Stable on standing—may form a creamy top layer
3. No odor, except what may be due to volatile fatty acids
4. Sterile, tend to resist putrefaction
5. Alkaline reaction
6. Fat 0.4 to 4.0 per cent
7. Specific gravity greater than 1.012
8. Protein content variable
9. Solids 7.04 per cent
10. Smear shows varying numbers of white cells with lymphocytes predominating
11. Removal of fat by addition of alkali and ether causes the fluid to clear

*After Brescia.

CAUSATION OF CHYLOTHORAX

The causes of chylothorax have been tabulated by McNab and Scarlett[8] (Table II). Trauma is the responsible factor in more than one-third of all cases reported. Severe coughing is the apparent cause in some cases. One of our patients suffered his first symptoms after a bout of violent coughing. Bruises to the thorax and fractures of the clavicles or ribs are sometimes responsible for the occurrence of chyle in the pleural space. Overvigorous attempts to resuscitate a newborn infant may result in rupture of the thoracic duct.[9] Nine cases of chylothorax due to gunshot wounds have been reported in the literature.[10, 11] Blast injuries both in war and in peace time may cause this condition. Injuries to the thoracic duct have occurred in the course of surgical operations. Chylothorax has been reported following lumbar sympathectomy[12, 13] and after operations in the supraclavicular fossa.

Chylothorax of nontraumatic origin occurs most frequently in association with malignant disease, especially when metastatic involvement occurs. Invasion of the thoracic duct and its radicles, or the subclavian or innominate veins, or even the superior vena cava is fairly common. Such metastatic involvement is encountered most often in cases of primary carcinoma of the stomach[14, 15] or of neoplasms of the lympho-

TABLE II

CAUSES OF CHYLOTHORAX*

1. Trauma
   - External violence
     - Closed trauma—no external wounds or fractures
     - Trauma with fractured ribs, clavicles, or vertebrae
     - Gunshot wounds
     - Stab wounds
   - Operative wounds
     - Complete severance of the duct
     - Section of one or more terminals
2. New growths and granulomas
   - Carcinoma, lymphosarcoma, tuberculous glands
3. Thrombosis of the left subclavian vein
4. New growths of the duct itself
5. Perforating lymphangitis
6. Aneurysm of the duct
7. Cirrhosis of the liver
8. Filaria

*After McNab and Scarlett.

blastoma group. Tuberculosis has been considered the responsible factor in many cases. Tuberculosis of the lymph nodes of the mediastinum and the retraction caused by healing tuberculous lesions of the lung have been held responsible. In one of our cases tuberculous paravertebral abscess was the apparent cause. The ability of filarial parasites to obstruct the lymphatic circulation is well known. Perhaps additional cases of chylothorax will be reported as the result of the prevalence of filarial infections in military personnel.

It is of interest that experimental ligation of the thoracic duct fails to produce chylothorax.[16, 17] This is probably explained by the numerous collateral vessels which join the thoracic duct with venous channels. It is possible to produce chylothorax in about 50 per cent of cases when the superior vena cava is ligated.[17, 18] Ligation of the superior vena cava raises the venous pressure in nearly all the branches into which the lymphatic vessels can empty.

Chylothorax may occur in either pleural space or sometimes is bilateral. Right chylothorax is much more common than left chylothorax. In our series there were six instances of involvement of the right side and three instances in which the left side was affected. In two of the cases of effusion of the right side fluid was detected in the left side in lesser amounts. It is not surprising that right chylothorax is the more common since the duct traverses most of the thorax on the right side of the vertebral column. Injuries of the upper part of the thorax are likely to give rise to chylothorax on the left side while injuries in the lower two-thirds of the thorax may result in chylothorax on the right.

CLINICAL FEATURES OF CHYLOTHORAX

The signs and symptoms of chylothorax secondary to trauma are listed in Table III. The development of chylothorax in nontraumatic cases is

TABLE III

SIGNS AND SYMPTOMS OF CHYLOTHORAX SECONDARY TO TRAUMA*

1. Delay in onset—two to ten days after trauma
2. Dyspnea—onset rather sudden
3. Mild shock or even collapse
4. Intrathoracic accumulation of chyle (massive)
5. Reappearance of chyle in thorax after thoracentesis
6. Inanition, oliguria, thirst, emaciation
7. Healing of severed duct in two to three weeks if patient survives

*Modified after Whitcomb and Scoville.[12]

far less dramatic than in cases due to trauma. The thoracic duct is an extrapleural structure and, unless trauma causes a perforation of the pleura, the first accumulation of chylous fluid must take place behind the pleural space. Retropleural chylomas have been described by Brown[19] and by Nowak and Barton.[20] This extrapleural collection of chyle must then rupture into the pleural space. This seems to provide the best explanation for the delay in onset of symptoms following trauma to the thorax. Dyspnea and shock are the predominant initial symptoms. These symptoms may be severe and relief may be equally dramatic after thoracentesis. Usually there is rapid reaccumulation of the pleural fluid, necessitating frequent aspiration of the pleural space. Dehydration and emaciation are likely to occur. When there is rapid reaccumulation of pleural fluid the patient's condition becomes steadily worse and death may occur in a few days. If the fluid reappears slowly and thoracentesis is necessary only at infrequent intervals, the patient may live for weeks or months. In approximately 50 per cent of reported cases of traumatic chylothorax, healing of the severed duct apparently has taken place and the patients have recovered.[21] Chylothorax among children has been reported in about twenty-five cases.[7] In about one-half of these the rupture of the duct was apparently spontaneous. The mortality rate in these cases was also about 50 per cent.

Chylopericardium and chylous ascites may occur in association with chylothorax. Chylopericardium is rare, as only four cases have been reported. Chylous ascites is more common than chylopericardium and may occur either with chylothorax or independently. In one of our cases chyloperitoneum was a late complication of chylothorax. Chylous ascites may be the result of surgical trauma to the cisterna chyli.

In cases in which frequent aspiration of chyle from the pleural space is required, peripheral edema is likely to occur. This edema is best explained on the basis of low serum proteins. Much protein and fat may be lost in the chyle and serum protein values are almost invariably lowered in those cases in which frequent thoracentesis is required.

DIAGNOSIS OF CHYLOTHORAX

Chylothorax should be suspected when pleural effusion is present after injuries to the thorax or when there is no obvious explanation for

pleural fluid in nontraumatic cases. The diagnosis of chylothorax is made by examination of the aspirated pleural fluid (Table I). The fluid is usually milky white in appearance. When it is dropped on a glass slide, the oily appearance may be detected. If it is allowed to stand in a test tube, a creamy layer may develop at the top of the tube. Sudan III will readily bring out the fatty nature of the fluid. The specific gravity is in excess of 1.012. A cell count will show a large number of cells with lymphocytes predominating. If the fluid is alkalinized and shaken with ether, it will clear. Chemical examination will show that the total fat content varies from 0.4 per cent to 4 per cent. The usual bacteriologic and pathologic studies of pleural fluid do not assist one in making the diagnosis of chylothorax. Whenever milky effusions are encountered the diagnosis should be made readily by staining the fluid with sudan III, by shaking the fluid with ether, and by determination of the fat content.

TREATMENT OF CHYLOTHORAX

Brown[19] successfully drained a retropleural chyloma, that is, the extrapleural accumulation of chyle that precedes chylothorax. Open explorations of the thoracic cavity in the hope of surgically closing a ruptured thoracic duct have not met with success.[12, 21]

Medical measures are directed to maintain the bodily nutrition and to replace the serum proteins and fats lost in the chyle. A high carbohydrate, high protein diet has been advised in these cases. A number of instances have been recorded in which aspirated chyle has been reintroduced into the venous circulation in the hope of preventing peripheral edema.[6, 10, 13, 21-24] Sodium citrate or heparin must be used as an anticoagulant. Some beneficial results have been reported. An occasional death has resulted from the intravenous infusion of chyle.[12] Solution of acacia or blood plasma given intravenously is a safer method of restoring normal osmotic pressure in the blood.

The treatment of chylothorax has not been satisfactory. Thoracentesis is necessary for the relief of acute respiratory embarrassment. It is not always desirable to try to keep the pleural space empty by repeated thoracentesis, as the fluid tends to reaccumulate rapidly. In about 50 per cent of cases of chylothorax of traumatic origin, healing of the ruptured thoracic duct takes place in the course of a few weeks.[12] These patients make a complete recovery. In some reported cases an attempt has been made to close the rupture of the thoracic duct by the introduction of foreign substances into the pleural space. Successful results have been reported after the introduction of sterile broth[10] or of gomenol[11] into the pleural space. One patient who had right chylothorax of traumatic origin was cured after interruption of the phrenic nerve on the affected side.[20] The elevation of the diaphragm presumably closed a perforation of the thoracic duct in the lower part of the thorax.

Roentgen therapy has been used in the treatment of the nontraumatic forms of chylothorax. It has been of most value in cases in which chylothorax was secondary to malignant disease, especially lymphoblastoma.[24] The prognosis in nontraumatic cases is generally poor, as the underlying disease is usually serious. In a number of cases of chylothorax no obvious etiologic factor can be demonstrated. It is probable that some trauma is responsible for such instances. However, the possibility of malignant disease must always be kept in mind.

ANALYSIS OF MAYO CLINIC EXPERIENCE

As previously stated, nine patients who had chylothorax have been seen at the Mayo Clinic. Seven of these patients have been seen very recently. Of these nine patients, five were men and four were women. Trauma was the apparent cause in only one case. In three cases malignant disease was the obvious cause of the chylothorax. Malignant disease was suspected but not proved in one case. Tuberculosis of the vertebrae with paravertebral abscess was responsible for hydrothorax in one case. In three cases we have been unable to find any explanation for chylothorax.

REPORT OF CASES

The important features regarding each of these cases are as follows:

CASE 1.—A man, aged 44 years, was seen at the clinic in 1920. Right chylothorax was present and the fat content of the fluid was 1.2 per cent. There were enlarged lymph nodes in the left supraclavicular region and on the rectal shelf which were characteristic of metastatic malignant disease. The patient died three months later.

CASE 2.—A man, aged 60 years, registered at the clinic in July, 1936. He presented the typical clinical picture of Pott's disease with roentgenographic evidence of destruction of the ninth and tenth thoracic vertebrae. Fluid was present in the right pleural space. The fluid was oily and on microscopic examination contained many crystals of cholesterol. Guinea pigs inoculated with this fluid died of tuberculosis. The patient was sent to a tuberculosis sanatorium.

CASE 3.—A married woman, aged 53 years, was seen in December, 1942. A milky fluid was aspirated from the left pleural space. Chemical analysis of the fluid showed 0.7 per cent of fat. At necropsy a large mass of enlarged lymph nodes was found in the superior mediastinum. Microscopic examination showed lymphosarcoma.

CASE 4.—A married woman, aged 58 years, was seen in March, 1943. Hydrothorax of the left side was found, but no explanation could be given for this finding. Thoracentesis revealed a milky fluid containing droplets of oil. The cause of the chylothorax was not clear.

CASE 5.—A man, aged 25 years, was seen in February, 1943. For two years he had had dyspnea due to a right pleural effusion. Many thoracenteses had revealed milky fluid. The fat content was slightly more than 2 per cent. Although the diagnosis has not been clear, roentgen therapy has had beneficial results.

CASE 6.—A woman, aged 40 years, has been a patient at the clinic since 1939 because of dyspnea. Chyle has been aspirated from the right pleural space on many occasions and more recently from the left pleural space and the abdominal cavity. The fat content of the pleural fluid was 2.7 per cent and of the peritoneal fluid 4 per cent. Recently edema of the extremities has developed. No explanation has been found for the chylothorax but malignant disease has been suspected.

CASE 7.—A man, aged 41 years, was seen at the clinic in May, 1943. Pleural effusion of the right side had developed after a severe coughing spell. Aspiration revealed that chyle was present in the pleural space. The patient has had repeated thoracenteses since the onset. Analysis of fluid showed fat content to be 2 per cent. At the date of writing, the patient seemed to be gradually improving. The etiologic factor was not certain.

CASE 8.—A married woman, aged 67 years, was seen in November, 1943. She complained of swelling in the left supraclavicular region, left thoracic wall, and left arm and hand. This subsided but was followed by swelling of the abdomen and lower extremities. Roentgenograms of the thorax disclosed a bilateral pleural effusion, more marked on the right than on the left side. Right thoracentesis revealed a milky fluid. Chemical analysis showed 2 per cent fat. Pathologic examination of the fluid showed carcinoma grade 4 (Broders' method). This was confirmed by biopsy of a cervical node.

CASE 9.—A man, aged 51 years, was seen at the clinic in September, 1943. For twelve years he had experienced gradually increasing edema of the lower extremities and swelling of the scrotum and the abdomen. More recently he had been dyspneic on exertion. On admission he had marked edema of the lower extremities and scrotum, evidence of fluid in the left pleural space, ascites, and a large liver. The concentration of protein was 3.8 mg. per 100 c.c. of serum. Thoracentesis revealed a milky fluid containing 0.64 per cent fat. Under hospital management and treatment with potassium nitrate and intravenously administered solutions of acacia, the edema subsided. No definite diagnosis could be made. Numerous laboratory studies, including tests of renal and hepatic function and careful evaluation of the cardiac status, failed to reveal any other cause for the edema. Reduced osmotic pressure caused by lowered serum proteins was undoubtedly the cause of the edema and

ascites. However, we did not feel that we could attribute the reduction of serum proteins to the disturbance of the circulation of chyle in this case.

COMMENT

It is our impression that many instances of chylothorax occur which are not recognized. Chylothorax must be suspected whenever a milky fluid is obtained at thoracentesis, especially if no obvious reason for pleural fluid is present. The simple tests which have been outlined and chemical analysis of the fluid will confirm a diagnosis of chylothorax. War injuries, especially gunshot wounds of the thorax and blast injuries, may be a frequent cause of chylothorax.

SUMMARY

Chylothorax may be of traumatic or nontraumatic origin. The clinical syndrome is quite different in the two varieties. The symptoms of chylothorax following injury to the thorax present a characteristic pattern, but chylothorax of nontraumatic origin is most often insidious in onset.

The diagnosis of chylothorax rests chiefly on examination of the pleural fluid. The demonstration of excess fat in pleural fluid is pathognomonic of chylothorax. Chylous ascites and, more rarely, chylopericardium may occur in association with chylothorax.

The prognosis of chylothorax is generally poor. In the nontraumatic forms, the serious nature of the primary process, most frequently malignant neoplastic disease, is responsible for the fatal outcome. In cases in which the course is protracted the loss of protein in aspirated chyle results in lowered osmotic pressure of the blood and extensive edema may occur. The mortality rate of chylothorax of traumatic origin is about 50 per cent.

There is no satisfactory method of treatment for chylothorax. Inasmuch as 50 per cent of patients who have chylothorax of traumatic origin recover spontaneously, it is difficult to evaluate the various methods of therapy. Treatment of chylothorax of nontraumatic origin is usually unsatisfactory because of the serious nature of the underlying disease.

Nine cases of chylothorax have been recognized at the Mayo Clinic. Seven of these nine cases were under observation at the clinic in the period from December, 1942, to November, 1943, inclusive.

It is felt that chylothorax occurs much more frequently than is generally recognized. Careful observation of pleural fluid and a few simple tests are required to make the proper diagnosis.

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ANOMALOUS PULMONARY VEINS

Report of a Case

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A GREAT many different types of anomalies of both the pulmonary arteries and the pulmonary veins have been described in the literature. The majority of these articles have appeared in the anatomical journals. Because of the advancement of thoracic surgery, these anomalies have become of clinical significance.

The first case of anomalous pulmonary veins which we were able to locate in the literature was that of Winslow (1739) in which the upper lobe of the right lung was drained by a single vein into the superior vena cava. Brody analyzed 102 similar cases that he was able to collect from the literature up to 1942. He classified them into two groups: (1) those which open in part, directly or indirectly, into the right atrium and (2) those which drain entirely into the right atrium. The most common type of partial drainage into the right artium was that associated with one or more anomalous veins through which blood passed from the upper lobe of the right lung into the superior vena cava. According to the table given by Brody, 44 per cent of the patients exhibited this anomaly. The point of entrance varied widely. The other frequent sites of drainage were directly into the right atrium in 27 per cent, and into the left innominate vein in 21 per cent of the cases. Occasional sites of drainage were: coronary sinus, inferior vena cava, azygos vein, portal vein, and left subclavian vein. The right pulmonary veins were involved about twice as frequently as the left.

The cases in which all the pulmonary veins enter the right atrium are not likely to be of surgical importance because these persons ordinarily die at birth or shortly thereafter, unless there is a compensating anomaly such as a patent ductus arteriosus, or a patent foramen ovale. Partial drainage of the pulmonary veins into the systemic circulation is, however, compatible with life as shown by Brody's report of sixty-eight cases in which more than 74 per cent reached maturity. He further states that apparently a minimum of 50 per cent of the aerated blood must enter the left atrium in order to maintain an adequate oxygen level in the systemic circuit. On the basis of a survey of the reported cases it seems probable that when as much as 75 per cent of the pulmonary return goes to the left artium, there are no recognizable cardiorespiratory symptoms.

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The case to be reported here was that furnished by the body of a 48-year-old man dissected in the anatomical laboratories of the University of Michigan. The entire upper lobe of the right lung was drained by three tributaries converging into a common trunk, 15 mm. in diameter, which emptied into the superior vena cava (Fig. 1). This aberrant trunk passed slightly superior and anterior to both the superior branch of the pulmonary artery and the right bronchus. It entered the superior vena cava just inferior to the entrance of the azygos vein and about 3 cm. superior to the right atrium. The upper lobe of the right lung was congested and dark red in color. There was an incomplete

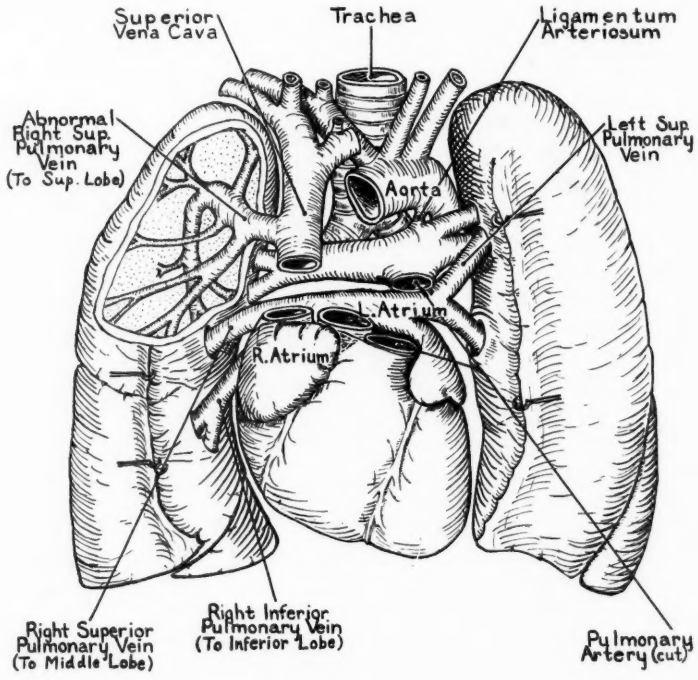

Fig. 1.

transverse fissure but the rest of the lung was entirely normal. The superior pulmonary vein, which was approximately 50 per cent smaller than normal, drained only the middle lobe, and the inferior pulmonary vein drained the inferior lobe of the right lung. Both these veins entered the left atrium in the normal fashion. There were no gross connections between any of the aberrant vessels and the right superior pulmonary vein, either inside or outside the lung. The pulmonary veins on the left showed no abnormalities. The right side of the heart was not disproportionately enlarged, and the heart as a whole showed

only slight enlargement. No anomalies were found in the heart or in the rest of the body.

This patient had been in an institution for fifteen years prior to death because of central nervous system syphilis. Repeated physical examinations failed to demonstrate any cardiorespiratory abnormalities. X-rays of the chest were essentially negative. There was no history of dyspnea or cyanosis with moderate activity, even during his last years. On the morning of his death he suddenly slumped over in a chair, screamed with pain, became pale and cyanotic, and perspired freely. His pulse was irregular and poor in quality. He died approximately fifteen minutes later. We were unable to find from our dissection any definite cause for his sudden death.

The interpretation of the origin of these anomalies involves details of the embryonic circulation which are not ordinarily emphasized in the embryologic textbooks. In the journal literature, however, may be found detailed studies of the plexus of small vessels surrounding the developing mediastinal structures. In this part of the embryonic body there is at first a meshwork of small vascular channels which are drained primarily into the anterior cardinal veins. Secondarily new vessels develop which divert the return from part of this primitive plexus directly back of the heart. These new vessels will form the pulmonary veins. Flint and Auer state that these young pulmonary veins can first be recognized draining into the sinus venosus. Usually, however, these vessels are not sufficiently well formed to be readily recognizable until the sinus venosus has been crowded to the right of the developing interatrial septum primum. On this basis Brown and Evans speak of the developing pulmonary veins as entering directly into the left atrium. From this primitive vascular plexus, channels develop which subsequently become arteries or veins. Brown subdivides the plexus around the lung bud into two parts: (1) from its ventral and caudal portion a pulmonary part associated with respiratory function and (2) from the dorsal and cephalic portion a bronchial part concerned with nutrition of the stroma of the lungs. The communications between these vascular channels are greatly reduced with further development of the pulmonary vessels. Even in the normal adult, however, there remains the tendency for some of the smaller arterioles of the bronchial system to communicate with the pulmonary capillaries, and to have their blood drained by the pulmonary rather than by the bronchial veins. Anomalous drainage of the pulmonary circuit to the right side of the heart can be attributed to the persistence of some of the primitive connections of the pulmonary part of the primordial plexus with the anterior or common cardinal veins. With the conversion of these vessels on the right to form the superior vena cava there are established conditions such as those in the case described here.

From a clinical surgical standpoint, the venous abnormalities described in this article are important in connection with the operations of pulmonary lobectomy and total pneumonectomy. The surgeon should be aware of the occurrence of such anomalies. During the dissection of the pulmonary hilum in preparation for the ligation of the pulmonary vessels, he would almost certainly recognize and ligate any abnormal vein passing between the lung and mediastinum, even though he failed to appreciate the physiologic significance of the vein. Whether a proper clinical diagnosis of a case in which such a vein or veins is the cause of inadequate aëration of the systemic blood will ever be made and lead to the therapeutic ligation of one or more of the veins remains for the future to determine. We have seen no record of such a diagnosis having been made during life.

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Notice

The Twenty-Fifth Annual Meeting of the American Association for Thoracic Surgery, which was originally scheduled for 1942, will be held May 5 and 6 in Chicago, Ill. Headquarters will be at the Drake Hotel. The American Surgical Association will meet at the same place on the two preceding days and members of the two organizations are invited to attend all sessions.

All persons desiring to present papers at the meeting are urged to send in their requests as soon as possible. The requests should be accompanied by short but informative abstracts of the proposed papers, and directed to the Secretary, Lt. Col. Richard H. Meade, Jr., Kennedy General Hospital, Memphis 15, Tenn. It will be impossible to consider any applications after April 1, 1944.

Room reservations should be made at once. Address all such requests to Mr. W. M. Cowan, Manager, Room Reservation Department, Drake Hotel, Chicago, Ill., and state that the object of the visit is attendance at the meeting.

Richard H. Meade, Jr.
Secretary.